A STUDY OF THE CLINICAL PROFILE OF PATIENTS WITH MYOCARDIAL INFARCTION

Dissertation submitted to

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

CHENNAI- TAMILNADU

In partial fulfillment for the Degree of

DOCTOR OF MEDICINE

BRANCH I –M.D., (General Medicine)

APRIL-2015

DEPARTMENT OF MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 627011

TAMILNADU

CERTIFICATE

This is to certify that the Dissertation entitled "**A STUDY OF THE CLINICAL PROFILE OF PATIENTS WITH MYOCARDIAL INFARCTION"** submitted by **Dr.E.SARAVANAN** to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for M.D.(Branch-I) General Medicine Examination to be held on April 2015 is a bonafide work carried out by him under my guidance and supervision. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

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CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 414/GM/2013/29.03.13

PROTOCOL TITLE: A Study of the Clinical Profile of Patients with Myocardial Infarction

NAME OF PRINCIPAL INVESTIGATOR: E.Saravanan

DESIGNATION OF PRINCIPAL INVESTIGATOR: Post Graduate Resident

DEPARTMENT & INSTITUTION: Department of General Medicine, Tirunelveli Medical College

Dear Dr E.Saravanan, *the Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on* 29.03.2013.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed

STANDS APPROVED UNDER SEAL

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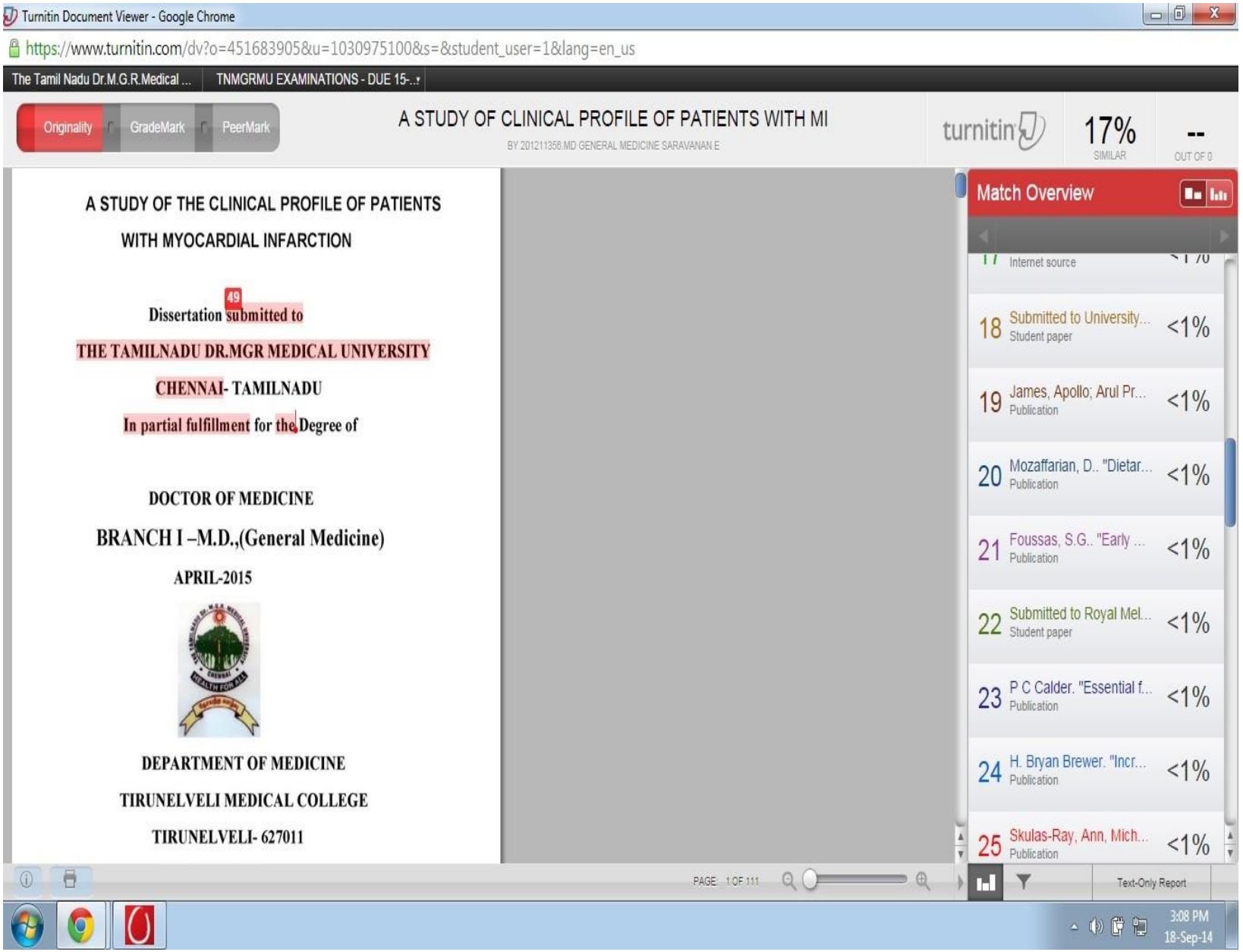
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DECLARATION

I**, Dr.E.SARAVANAN**, solemnly declare that the I carried out this work on **"A STUDY OF THE CLINICAL PROFILE OF PATIENTS WITH MYOCARDIAL INFARCTION"** at Department of General Medicine, Tirunelveli Medical College and Hospital during the period of August 2013 to August 2014.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the rules and regulations for the MD Degree Branch I (General Medicine) Examination.

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

Place: TIRUNELVELI

Date:

DR.E.SARAVANAN,

POST GRADUATE,

M.D. GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

ACKNOWLEDGEMENT

At the outset I wish to thank our Dean Dr.THULASIRAM, MS, for permitting me to carry out this study in our hospital.

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I also thank the Departments of Microbiology and Biochemistry for the laboratory support to this study.

No words of gratitude will be enough to thank my parents for their unconditional support and encouragement at each step in my way.

Last but not the least, I sincerely thank all the patients who cooperated with me by participating in the study.

CONTENTS

A STUDY OF CLINICAL PROFILE OF PATIENTS WITH MYOCARDIAL INFARCTION

ABSTRACT:

BACKGROUND:

Cardiovascular Diseases (CVD) is a leading cause of non communicable morbidity and mortality in India. By the year 2030 CVD is projected to be the leading cause of death worldwide. One of the most important advances in cardiovascular research was the identification of risk factors associated with cardiovascular diseases with subsequent treatments developed and rigorously tested to modify the risk factors with the goal of preventing CVD. Although 80% of global burden of cardiovascular diseases occurs in low and middle income countries, knowledge of importance of risk factors is largely derived from developed countries. The effect of factors on risk of coronary artery disease in most regions of the world including developing countries like India remains largely unknown. Various studies have been conducted to identify risk factors associated with Cardiovascular Diseases**.**

AIM:

To study the symptoms, clinical profile, risk factors, complications and outcome in predominantly rural Indian population.

MATERIALS AND METHODS:

An observational cross sectional study involving 200 patients admitted with a diagnosis of ST Elevation Myocardial Infarction in Tirunelveli Medical College between August 2013 and August 2014. Data was collected based on Inclusion and Exclusion criteria.

INCLUSION CRITERIA:

All patients admitted to our hospital with a diagnosis of MI. The final diagnosis of MI will be based on the following criteria:

1. Ischemic chest pain for atleast 30 minutes

2. ECG evidence of myocardial injury:

0.1 mv or more ST segment elevation in 2 contiguous limb leads or

Development of pathological Q waves

3. An increase in serial CPK-MB or serial Troponin T or I

EXCLUSION CRITERIA:

Patients admitted with MI who refused to give consent for the study. Patients who are known cases of CKD, OLD MI, post angioplasty, post CABG patients.

The outcome of the patients at the end of one week was studied using TIMI (Thrombolysis In Myocardial Infarction) Risk Score calculated at the time of admission. The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2010) developed by Centre for Disease Control, Atlanta, USA.

RESULTS:

Among the 200 patients, 91% were discharged alive and 9% expired. The most common cause of death was cardiogenic shock. Among the 18 patients who expired, 14 patients had a Thrombolysis in Myocardial Infarction (TIMI) risk score of 9 and above and the remaining four had a TIMI score of 5 to 8. Mortality was not seen in those with a TIMI risk score of below five. The association was statistically significant ($p<0.0001$). In a subgroup analysis, In Young MI (<45 years), 91.7% were smokers, 88.9% consumed alcohol, around 50% had positive family history; 47% were leading a sedentary life style and everyone

100% had low HDL. The percentage of smoking and alcoholism were higher in young MI group compared to overall study group and all the patients in the young MI group were male.

CONCLUSION:

Cardiovascular Diseases affect mainly economically productive age group in our country and has become a major public health burden. Smoking, Alcoholism, Diabetes, Hypertension and Dyslipidemia remain the most important modifiable risk factors. Patients with a higher TIMI Risk Score have a greater mortality.

KEYWORDS: Cardiovascular diseases, Myocardial Infarction, Thrombolysis In Myocardial Infarction.

INTRODUCTION

Cardiovascular Diseases is an important cause of morbidity and mortality in the developed as well as the developing world. By 2030, WHO predicts that 33%of the deaths occurring worldwide will be caused by Cardiovascular diseases. With the epidemiologic transition well and truly taking place humans have travelled a long way from the stage of pestilence and famine to the stage of obesity and inactivity. Due to these factors we face a host of challenges and the morbidity due to cardiovascular diseases is accelerating with each passing day. In India alone Cardiovascular diseases account for 25% of the total deaths.

In our country the CVD risk factors among the rural as well as the urban poor and middle class are on the rise. The demon of cardiovascular diseases is very much at our door step. This is a frightening scenario considering that India is home to almost 17% of the world's population. With a bulging population not to mention the bulging waistline of the populace the challenges that lie ahead of us are monumental to say the least. Like many other non-communicable diseases , cardiovascular diseases have a long latency and have numerous modifiable risk factors .

One of the important advances in cardiovascular research has been with regard to the identification of risk factors associated with cardiovascular diseases. Based on these risk factors treatment plans have been drawn and meticulously tested with the goal of altering the outcome.

The major chunk of the global burden of these diseases is from the low and middle-income countries like India. But the data regarding the importance of risk factors has been derived mainly from developed countries.

In a multifaceted country like ours, with various ethnicities, cultural and food patterns and above all with varied political outlooks on the aspects of health coupled with significant infrastructure limitations has effectively prevented us from having completely representative comprehensive surveys with regard to demography. The disease and death registration systems is in a state of shambles. This has been a big bane of our health care system and has prevented us from taking pre-emptive measures. With a health care system as frail as ours with numerous lacunae it becomes even more imperative to initiate cost-effective solutions.

Numerous studies have been conducted to highlight the importance of risk factors associated with Cardiovascular diseases. This study is based on risks seen in predominantly rural population in and around Tirunelveli.

AIMS OF THE STUDY

AIMS OF THE STUDY

To study:

1. The type of presentation(symptoms)

2. The type of MI

3. The relative incidence of risk factors

4. The Thrombolysis in Myocardial Infarction(TIMI) score

5. Outcome

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

CORONARY ARTERY ANATOMY:[1]

LEFT MAIN CORONARY ARTERY:

It arises from the superior portion of Left aortic sinus, just below the sino tubular ridge of aorta. The sino tubular ridge separates the Sinus of Valsalva and the smooth tubular portion of aorta.

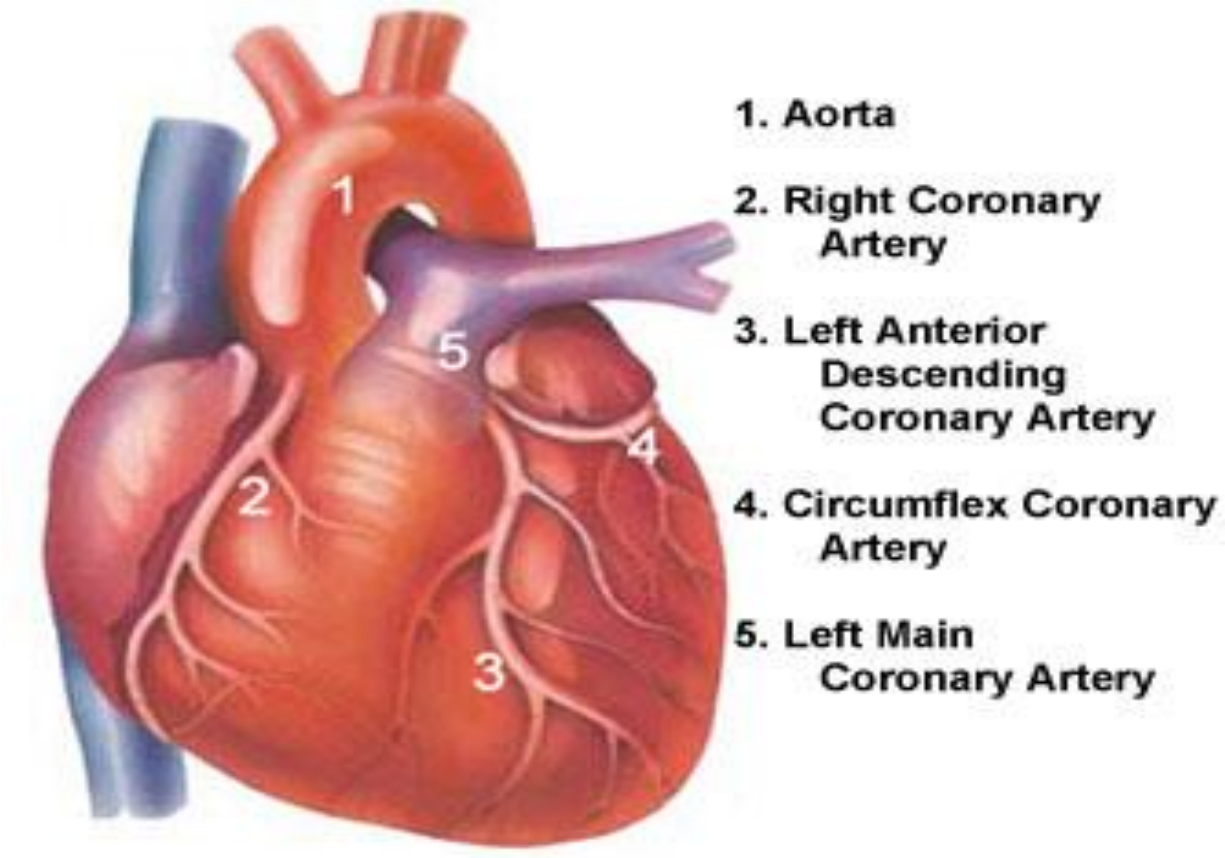

FIG.1 CORONARY ARTERIAL SUPPLY

LMCA has a variable length ranging from 10to 15 mm and an internal diameter that varies from 3 to 6mm. It runs behind Right Ventricular outflow tract and divides into Left Anterior Descending Artery (LAD) and Left Circumflex Artery. In a few cases, LMCA may be absent and there may be separateostia for LAD and LCX arteries. In some cases LMCA trifurcates into LAD , LCX and Ramus Intermedius.

LEFT ANTERIOR DESCENDING ARTERY:

It arises from the Left main Coronary Artery. This artery runs in the anterior interventricular groove along with the Great Cardiac vein towards the apex of the heart. It divides into septal and diagonal branches. Wide variation exists regarding the size and the number of septal perforators and diagonals. The Left Anterior Descending Artery supplies the anterior part of the septum via the septal perforators and the anterior, apical and the lateral walls of the left ventricle via the diagonal branches.

Commonly implicated in the majority of deaths due to Acute coronary Syndromes, it is commonly called “Widow makers’ artery”.

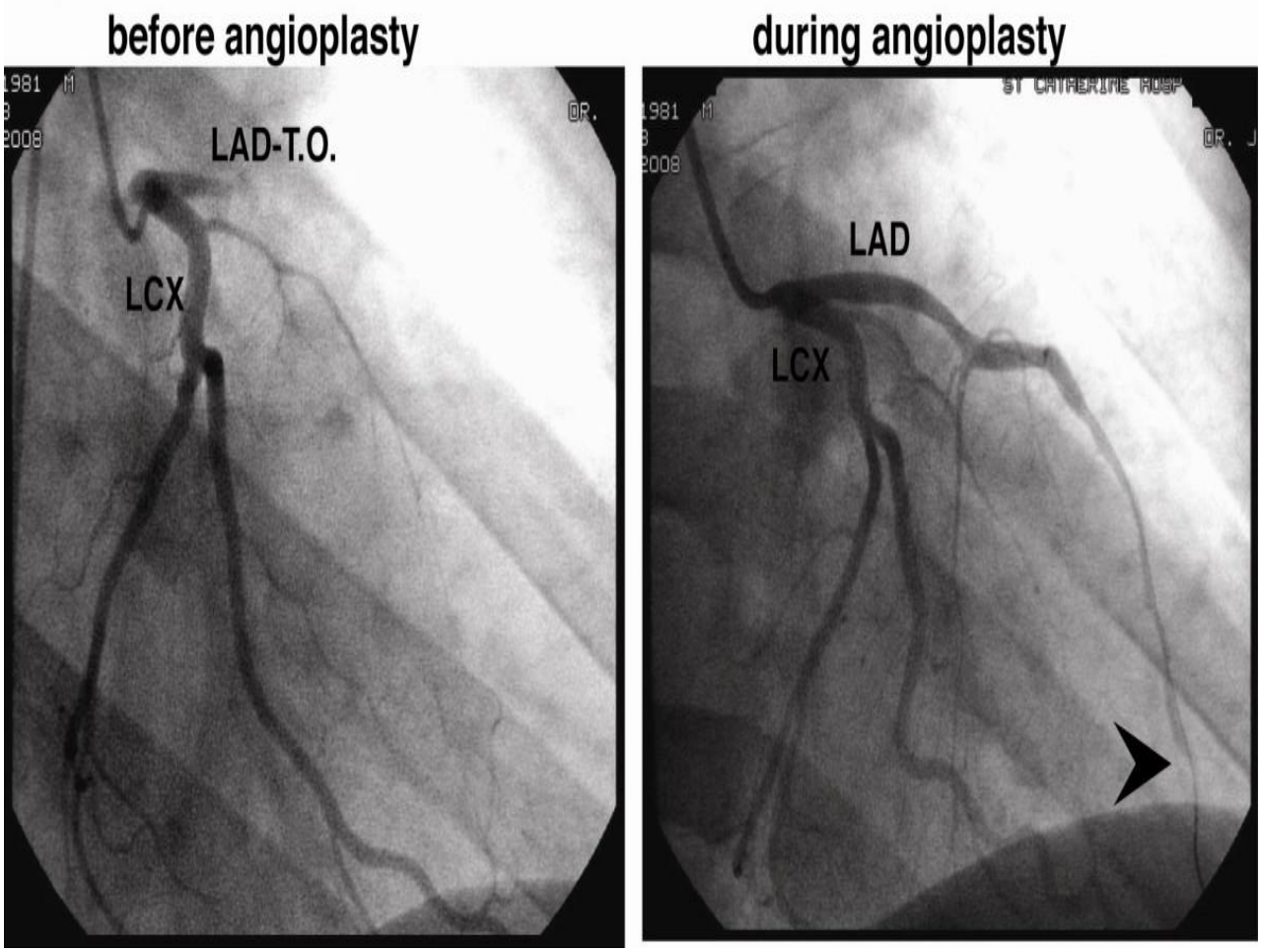

FIG. 2 TOTAL OCCLUSION OF LAD- ANGIOGRAPHY

LEFT CIRCUMFLEX ARTERY:

This artery originates from Left Main Coronary Artery and runs along posterior atrio ventricular groove toward inferior interventricular groove. It gives off large obtuse marginal branches. These Branches supply lateral free wall of left ventricle. In a few percentage of Individuals, Left Circumflex Artery gives off Posterior

Descending Artery (Left-Dominance). It also gives Left Atrial Circumflex branches that Supply posterior and lateral part of the left atrium.

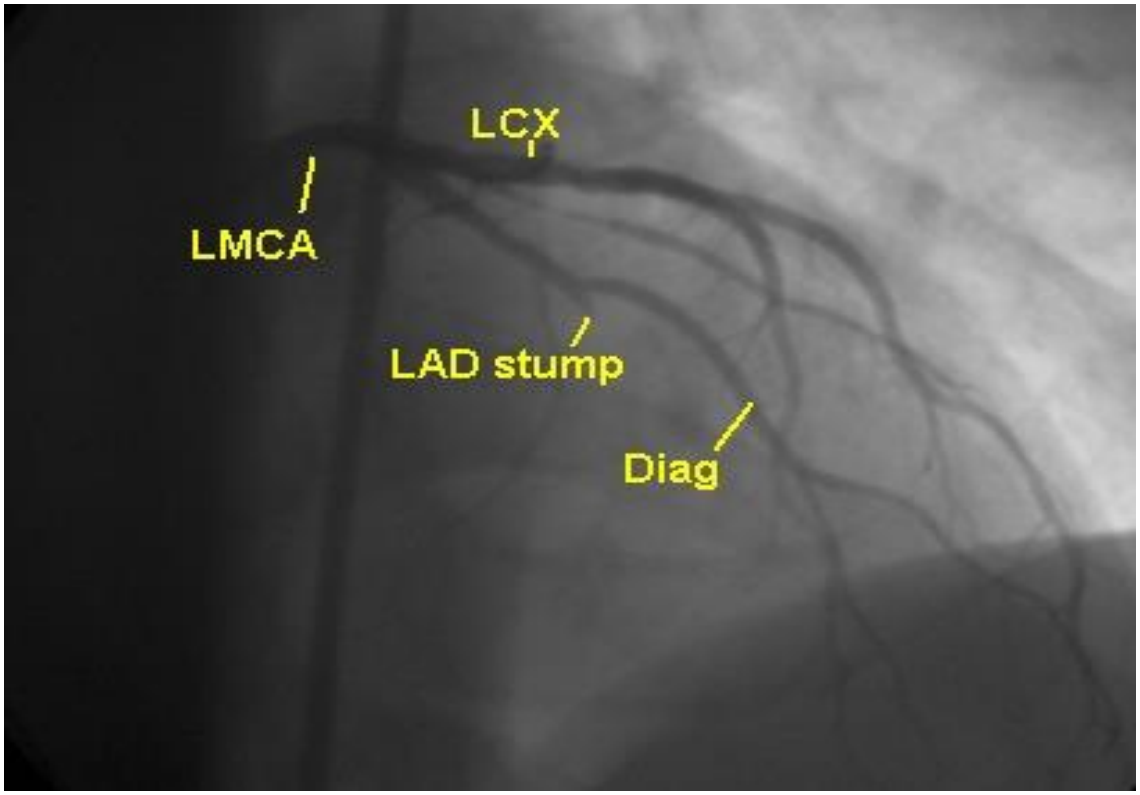

FIG.3 ANGIOGRAPHY- CORONARY ARTERIES

RIGHT CORONARY ARTERY:

This artery originates from right anterior aortic Sinus inferior to the origin of LMCA and runs along the posterior right atrioventricular groove. Its first branch is the conus artery,then it gives the Sinoatrial nodal artery in around 60% of the individuals. In the remaining the sinoatrial nodal artery arises from the Left Circumflex Artery in just under 40% of the cases with a few cases blood supply

coming from both the arteries. The Acute Marginal Branches arise from the mid portion of the vessel and supplies the anterior wall of the right ventricle. The RCA then gives posterolateral branches and continues as the posterior descending artery which supplies the lower part of the interventricular septum. Right dominance is seen in 85% of the individuals; that is RCA gives rise to the Posterior Descending artery and atleast one posterolateral branch. In the remaining 15%, in one half, Left Circumflex artery gives rise to the PDA and the posterolateral branches. In the other half ,PDA arises from the Right Coronary artery and the posterolateral branches from the Left circumflex Artery(co-dominant or balanced circulation). The Dominant artery usually gives rise to the Atrioventricular nodal artery which supplies the AV node.

CORONARY COLLATERAL CIRCULATION:

When a coronary vessel becomes totally occluded blood flow persists through collateral vessels. These channels develop when intracoronary pressure gradient occurs between source and recipient vessel. The extent and the function of coronary collaterals vary from patient to patient. In some, collaterals are well developed so that they are able to maintain perfusion at rest and also at sub maximal cardiac workloads[2]. Improved survival and a lower risk of cardiovascular events have been shown in patients with elevated distal coronary perfusion pressure due to recruitment of collaterals[3] . Repeated episodes of

coronary ischemia cause proliferation of coronary collaterals, a process called arteriogenesis[4]. When stenosis of the coronary vessel exceeds 70% the increase in endothelial shear stress in the pre-existing collateral vessels cause a Nitric Oxide dependent vasodilatation.

FIG.4 COLLATERAL CIRCULATION

But human randomized clinical trials to improve angiogenesis in mature coronary vessels has been highly disappointing[5]. But this may be because of the fact that improvements in functioning of the myocardium may be due to altered cell growth and repair rather than angiogenesis.[6] When Nitric Oxide synthesis is deficient, collaterals constrict and this can relieved by nitroglycerin.[7] This is similar to what occurs in the coronary resistance vessels.

CORONARY MICROCIRCULATION:

Individual coronary resistance arteries are a longitudinally distributed network and the mechanisms controlling the resistance in these arteries is variable. [8] These vessels dilate in a co-ordinated manner so that they are able to meet the demands of the distal vascular bed . This is done by shear stress mediated control of the blood vessels or through myogenic control.

Epicardial arteries with a diameter greater than 400 micron are mainly regulated by shear stress. The resistance vessels are divided into resistance arteries and arterioles. The arteries are regulated by shear stress and myogenic response whereas the arterioles are mainly controlled by local metabolic factors and they regulate the perfusion of coronary capillary bed. Average myocardial capillary density is around 3500/mm.square. The capillary density is considerably higher in the sub endocardium than the epicardium.

MYOGENIC CONTROL:

This is a mechanism by which vessels dilate in response to decrease in pressure and constrict when the pressure increases. It is due to the tone of smooth muscle [9]. It is believed to be due to the activation of the L type calcium channels. It occurs

mainly in arterioles<100 micron diameter and is an important mechanism in coronary auto regulation[10] .

Kuo and colleagues have shown flow-induced dilatation of coronary vessels [8,11]. This is mediated by NO and hence endothelium dependent. Endothelium Dependent Hyperpolarising factor ,an arachidonic acid metabolite primarily regulates epicardial conductance vessels[12]

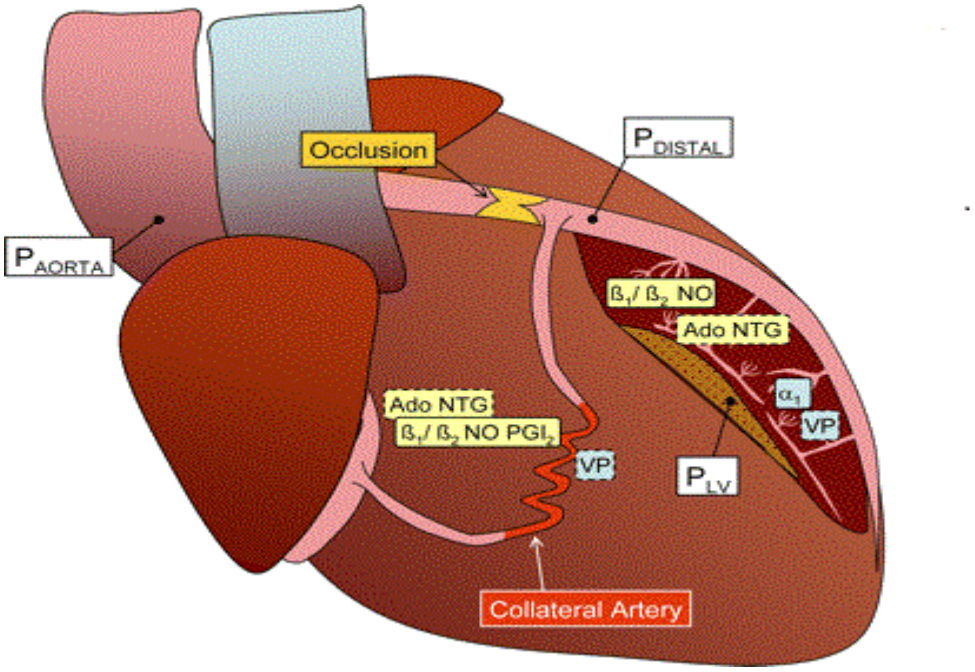

FIG.5 CORONARY MICROCIRCULATION

The resistance vessels are controlled by Nitric oxide[11]. EDHF may be upregulated during acquired disease states when Nitric oxide is deficient and serve as a compensatory mechanism assisting vasodilation[13].

VASCULAR BIOLOGY OF ATHEROSCLEROSIS:

THE NORMAL ARTERY:

The Normal Artery has three layers an innermost layer of tunica intima, middle layer of tunica media and the outer layer of tunica adventitia.

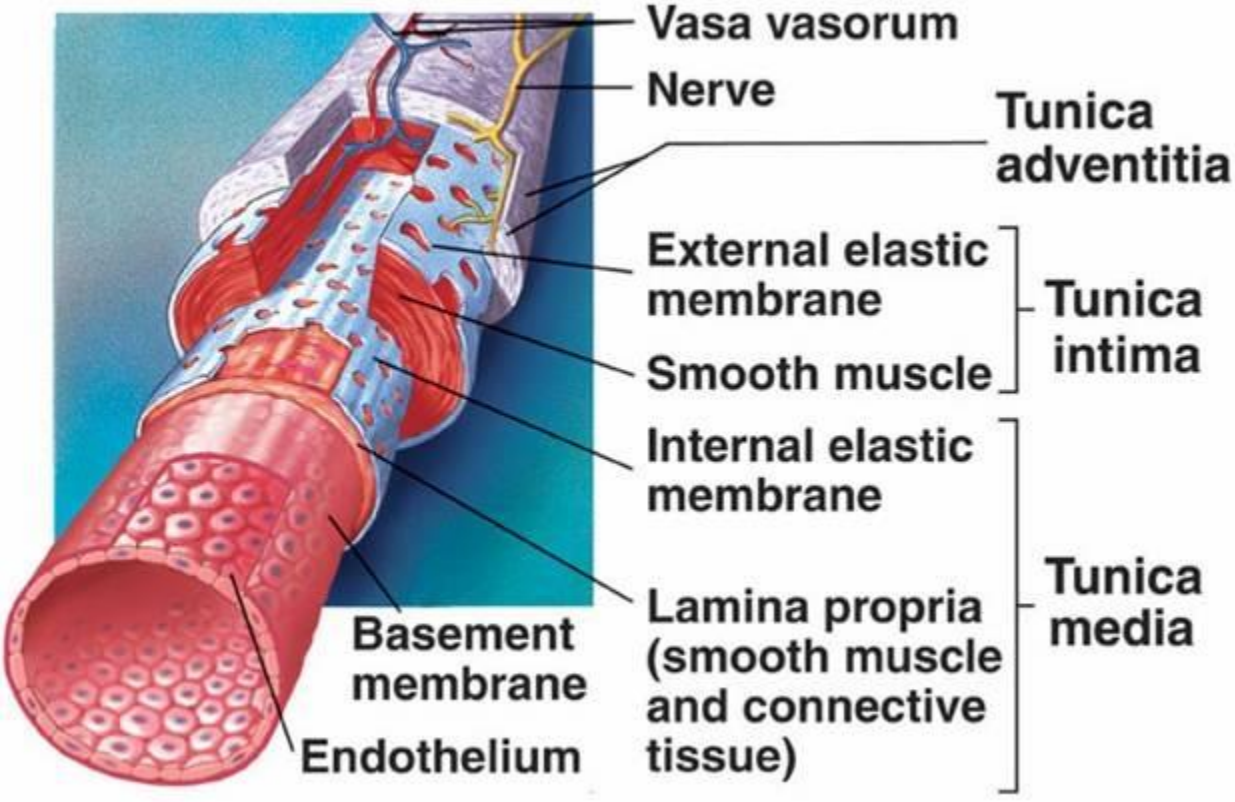

FIG.6 LAYERS OF ARTERIAL VESSEL WALL

TUNICA INTIMA:

The tunica intima is composed of a single layer of endothelial cells which rests on a basal lamina. The internal elastic lamina separates tunica intima and tunica media. The endothelial cells arise from precursor cells called Endothelial Progenitor Cells.

These cells help to repair areas of endothelial desquamation [14,15]. Older individuals have lowered levels of endothelial progenitor cells and hence less ability to repair any damage that occurs in the tunica intima [16]. New evidence has challenged the belief that Endothelial Progenitor cells populate murine atherosclerotic plaques [17].There is a variable expression of endothelial genes in different types of blood vessels. This is mainly regulated by the local environment[18]

TUNICA MEDIA:

The middle layer tunica media is composed of numerous layers of smooth muscle layers embedded in an extracellular matrix . The extracellular matrix is rich in elastin. This gives the tensile strength to the arteries. The external elastic lamina separates tunica media and tunica adventitia.

TUNICA ADVENTITIA:

This layer consists of loosely arranged collagen fibrils along with vasa vasorum and free nerve endings. The two most common types of cells seen are the fibroblasts and mast cells. Experimental evidence in animal models have shown that mast cells may well have an important in atheroma formation and also a role in pathogenesis of aneurysms[19].

ATHEROMA FORMATION:

The first step is accumulation of lipoprotein particles in the intimal layer followed by their aggregation[20].Oxidative stress, including products found in modified lipoproteins, results in the elaboration of various cytokines locally. The oxidative stress occurs in the form of oxidation, glycation, etc. The cytokines formed, increase expression of adhesion molecules for leukocytes that cause their attachment and chemoattractant molecules that direct their migration into the intima. The leukocytes thus move between endothelial cell junctions or sometimes penetrate through endothelial cells, a process called transcytosis. The leukocytes, on entering the artery wall in response to chemoattractant cytokines such as monocyte chemoattractant protein 1 (MCP-1) encounter stimuli such as macrophage colony-stimulating factor [21,22].These colony stimulating factors result

in increased expression of scavenger receptors. Scavenger receptors mediate the uptake of modified lipoprotein particles and promote the development of foam cells. T cells also tend to adhere to the endothelium and this effect is also mediated by leukocyte adhesion molecules.

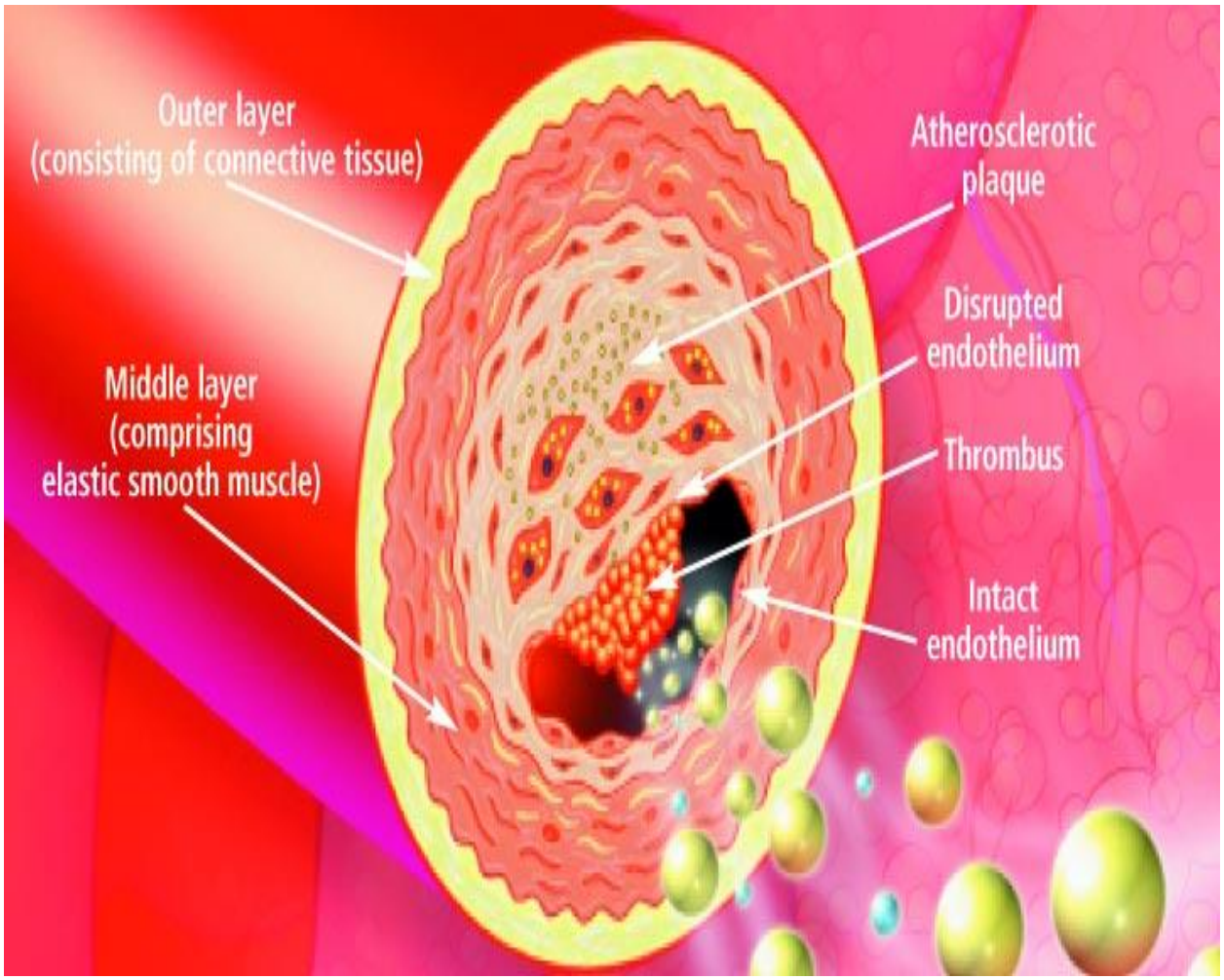

FIG.7 THROMBUS FORMATION

The commonly implicated leukocyte adhesion molecules are Vascular Cell Adhesion Molecule 1(V-CAM1),Inter cellular adhesion

Molecule 1(ICAM-1), E selectin, P selectin, etc. Macrophage foam cells are a source of mediators, such as further cytokines and effector molecules like hypochlorous acid, superoxide anion (O_2^-), and matrix metalloproteinases. These influences result in the migration of smooth muscle cells from the tunica media to the tunica intima. There is considerable variation between the smooth muscle cells found in the normal tunica media and those found in the intima of an evolving atheroma [23,24]. Emerging evidence has challenged the concept of migration of blood borne smooth muscle cells into the plaques [25].Further division of smooth muscle cells occur coupled with elaboration of extracellular matrix.

But an important point to note is that smooth muscle death also occurs hand in hand along with smooth muscle cell proliferation and migration. Interactions between the Fas ligand and the Fas expressed on the surface of smooth muscle cells coupled with the action of various pro inflammatory cytokines results in the death of smooth muscle cells. Thus the smooth muscle accumulation in the atheromatous plaque can be said as a finely done balancing act between smooth muscle cell replication and cell death. The extracellular matrix makes up the major volume of the plaque rather than the cells. It is composed of mainly collagen (types 1 and 3), proteoglycans like biglycan, aggrecan, decorin and versican. As the atherosclerotic plaques grow they develop their own blood supply. This angiogenesis is favoured by Vascular endothelial growth factor(VEGF),placental

growth factor (PIGF), certain forms of fibroblast growth factor and oncostatin M. This helps the plaque to overcome limitations in oxygen and nutrient supply and maintain its growth a mechanism similar to tumour angiogenesis[26].In later stages, calcification can occur and fibrosis continues, sometimes accompanied by smooth muscle cell death (including programmed cell death or apoptosis), yielding a relatively acellular fibrous capsule surrounding a lipid-rich core that may also contain dying or dead cells.

PATHOPHYSIOLOGY OF MYOCARDIAL ISCHEMIA:

The normal function of the heart muscle is supported by high rates of myocardial blood flow, oxygen consumption, and combustion of fat and carbohydrates (glucose and lactate). Under normal aerobic conditions, cardiac energy is mainly derived from fatty acids, which contribute around 60 to 90 percent for the synthesis of adenosine triphosphate (ATP). The rest of the energy (10 to 40 percent) comes from oxidation of pyruvate, formed from glycolysis and lactate oxidation. Almost all of the ATP formed comes from oxidative phosphorylation in the mitochondria; only a small amount of ATP (<2 percent) is synthesized by glycolysis. Approximately two-thirds of the ATP used by the heart goes to contractile

shortening; the remaining one-third is used by sarcoplasmic reticulum Ca^{2+} ATPase and other ion pumps.

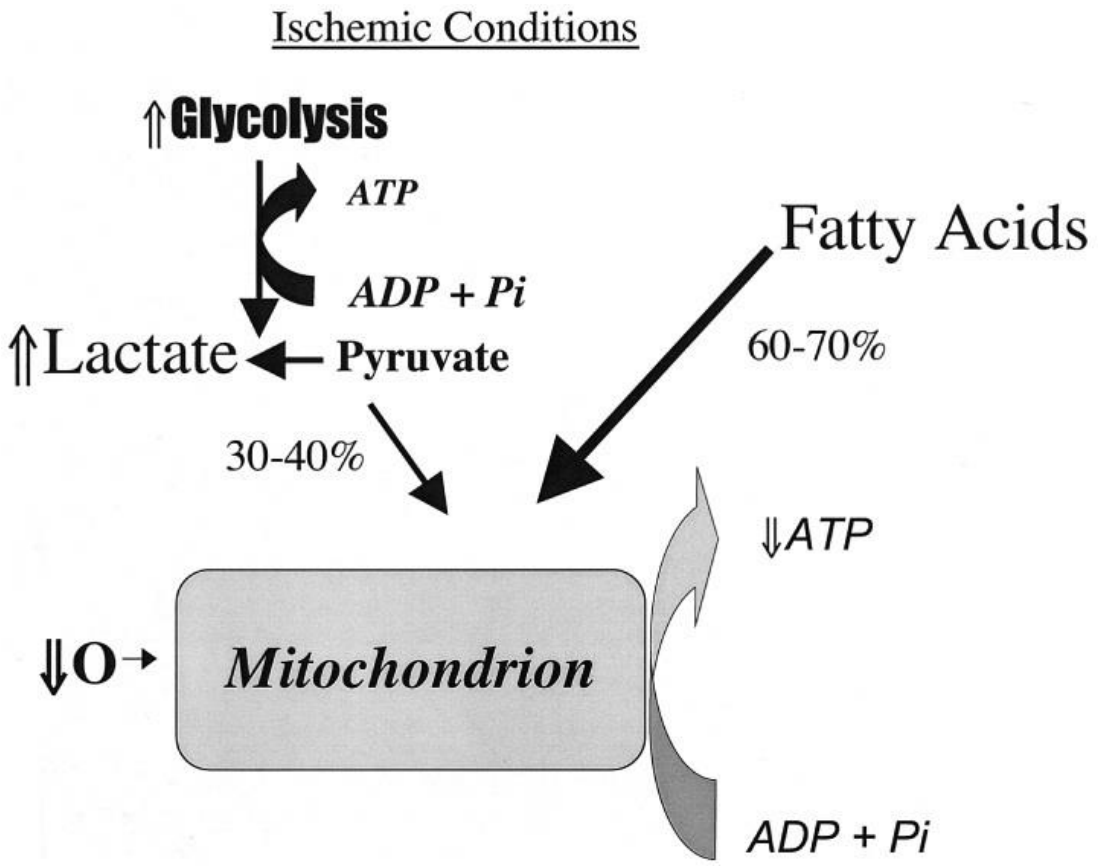

FIG. 8 PATHOPHYSIOLOGY OF MYOCARDIAL ISCHEMIA

When a major artery is suddenly occluded , a change in the metabolism occurs from aerobic or mitochondrial phosphorylation to anerobic glycolysis. This change occurs within a matter of seconds. This results in increased production of lactate and a fall in ph. This fall in ph leads to a greater ATP requirement in order to maintain calcium homeostasis [27]. Myocardial ischemia occurs due to an imbalance

between oxygen supply and demand. Biochemical abnormalities begin at the onset of ischemia, loss of myocardial contractility occurs within 60 seconds and the loss of viability occurs around 20 to 40 minutes following total coronary occlusion. Two zones of myocardial damage are seen: a central zone with no or very low flow and a zone of collateral vessels in a surrounding marginal zone. The survival of the marginal zone is dependent on two variables , one being the level of ischemia and the other being the duration of ischemia.

The extent of coronary collateral flow is one of the principal determinants of infarct size. Patients with well developed collaterals have low risk of developing an acute MI on total occlusion of coronary artery [28]. These collaterals seem to be well developed in younger individuals and also those who have experienced previous episodes of angina [29].

For the clinical diagnosis of acute myocardial infarction, the World Health Organization requires that at least two of the following three criteria be present:

(1) a history of chest pain or discomfort;

(2) a rise and subsequent fall in serum cardiac enzymes; and

(3) the development of electrocardiogram (ECG) abnormalities (new Q waves or ST-segment or T-wave changes) on serially obtained ECGs.[30]

Since the ECG lacks sufficient sensitivity and specificity to detect myocardial necrosis, the presence of myocardial injury is often dependent upon the release of cardiac-specific serum markers such as troponin T, troponin I, and CK-MB. It has been shown that infarct size generally correlates with the peak rise in serum CK-MB level.[31]

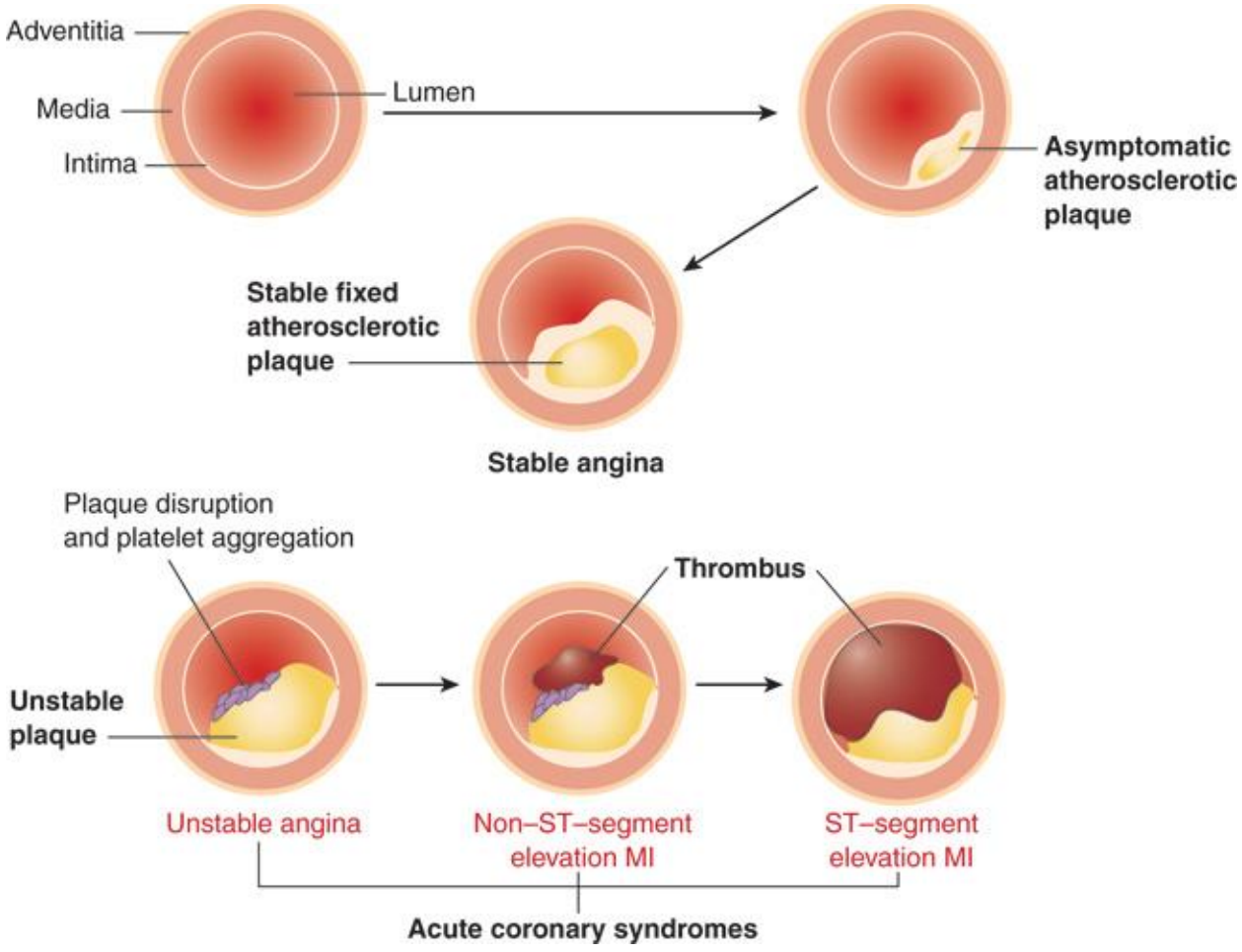

FIG.9 CLINICAL OUTCOME OF ATHEROMATOUS PLAQUE

WAVEFRONT PHENOMENON:

The area of myocardium that is necrosed, usually depends on the duration of coronary artery occlusion. This was first proved by Reimer and Jennings in an experiment using canine coronary artery which was occluded for variable duration. After only 15 min of occlusion, no infarct occurred. At 40 min, the infarct was sub endocardial, involving only the papillary muscle, placing 28 percent of the myocardium at risk. At 3 h following coronary artery occlusion and reperfusion, the infarct was significantly smaller compared with non reperfused permanently occluded infarct (62 percent of area at risk). The infarct size was greatest in permanent occlusion, becoming transmural and involving 75 percent of the area at risk[32] . In the dog model, it is impossible to achieve 100 percent infarction of area at risk because of species-related native collaterals. In humans, it has been shown that approximately 40 percent of patients with acute MI have a well-developed collateral circulation [29].

GROSS PATHOLOGICAL CHANGES:

The earliest change that usually occurs in the evolution of an acute MI is the pallor of the myocardium; this change usually occurs after 12 hours or later after the onset of irreversible ischemia. The detection of infarction can be enhanced by the use of tetrazolium salt solutions. These solutions form a colored precipitate on

gross section of fresh heart tissue in the presence of dehydrogenase-mediated activity. The tetrazolium salts [nitrobluetetrazolium (NBT) and 2,3,5-triphenyltetrazolium chloride (TTC)] are dyes that are sensitive to the presence of tissue dehydrogenase enzyme activity, which is depleted in the infarcted myocardium. It has been shown that myocardial infarct can be detected by NBT as early as 2 to 3 h in the dog and a little less in the pig because of poor collaterals. [33] Red (TTC) or blue color (NBT) will form only in the normal noninfarcted myocardium, thus revealing the pale, unstained infarcted region. In humans, the necrotic myocardium can be detected within 2 to 3 h after infarct by immersion of the fresh heart slices in a solution of TTC or NBT. TTC staining demonstrated a diagnostic sensitivity of 77 percent and a specificity of 93 percent compared with routine histology, with predictive values of positive and negative test of 81 and 91 percent, respectively.

Enhancement of pallor is seen 24 hours after the onset of ischemia. In this era of thrombolytic therapy, most in-hospital patients will have received tissue plasminogen activator, streptokinase, or IIb/IIIa inhibitors, which lyse the thrombus and restore blood flow into the area of infarction. Therefore, in a reperfused infarct, the infarcted region will appear red from trapping of the red cells and hemorrhage due to the rupture of the necrotic capillaries .

However, if there has been no reperfusion, the area of the infarct is better defined at 2 to 3 days, with a central area of yellow discoloration surrounded by a thin rim of highly vascularized hyperemia. At 5 to 7 days, the regions are much more distinct, with a central soft area and a depressed hyperemic border. At 1 to 2 weeks healing begins, with infiltration by macrophages as well as early fibroblasts at the margins. At the same time, the infarct begins to be more depressed, especially at the margins, where organization takes place, and there is a white border . The time taken for the infarct to completely heal may vary from as early as 4 to 6 weeks in small infarcts to as long as 2 to 3 months when the area of infarction is large. Healed infarcts are white from the scarring, and the ventricular wall may or may not be thinned (aneurysmal). In general, infarcts that are transmural and confluent are likely to result in thinning. But this is not the case with nonconfluent and sub endocardial infarcts which usually don't result in thinning.

LIGHT MICROSCOPIC CHANGES:

The earliest morphologic characteristic of MI that occurs is the hypereosinophilic myocyte.This change is usually seen between 12 to 24 h after onset of chest pain. The hypereosinophilia of the cytoplasm is best visualized by routine hematoxylin and eosin staining. The myocyte striations appear normal and some chromatin condensation may be seen in the nucleus. The area of infarction may show interstitial edema; however, this change is difficult to appreciate in human autopsy

hearts.This finding is better appreciated in animal models.The earliest characteristic change, the appearance of ‘wavy fibers’ is best seen in experimental animal models and they are believed to be the the result of stretching of the ischemic noncontractile fibers by the adjoining viable contracting myocytes.[34] Wavy fiber change is, however, nonspecific and occurs in the absence of ischemia, especially in the right ventricle. Neutrophil infiltration is present by 24 h at the border areas. As the infarct progresses between 24 and 48 h, coagulation necrosis is established, with various degrees of nuclear pyknosis, early karyorrhexis, and karyolysis. The myocyte striations are preserved and the sarcomeres elongate. The border areas show prominent neutrophil infiltration by 48 h.

After 3 to 5 days, the central portion of the infarct shows loss of myocyte nuclei and striations; in smaller infarcts, neutrophils invade within the infarct and fragment, resulting in more severe karyorrhexis (nuclear dust). Loss of myocyte striations is best visualised by Mallory's trichrome stain. Another stain that has been used to detect early areas of infarction is hematoxylin–basic fuchsin–picric acid. But this technique is not very reliable for the early detection (6 to 8 hours) of infarction in humans and hence not commonly used.

Immuno histochemical staining has also been used to study early changes of necrosis, but these tests which also include antibodies directed against creatine

kinase, ceruloplasmin, myoglobin, C-reactive protein, complement complex (C5b-9), fibronectin, and others—have also not been found to be useful. In the border areas of the infarct macrophage and fibroblast proliferation can be seen. By 1 week, neutrophils decline and granulation tissue is established, with neocapillary invasion as well as lymphocytic and plasma cell infiltration. Although lymphocytes may be seen as early as 2 to 3 days, they are not prominent at any stage of infarct evolution. Eosinophils may be seen within the inflammatory infiltrate but are present in only 24 percent of infarcts. There is phagocytic removal of the necrotic myocytes by macrophages, and pigment is seen within macrophages.

By the second week, fibroblasts become the most dominant cell type, but their presence can be seen as early as day 4 at the periphery of the infarct. Necrotic myocytes are continuously removed and the fibroblasts actively produce collagen and angiogenesis tends to occur in the area of healing. The healing continues to occur. Depending on the amount of cardiac myocytes necrosed, the healing may be complete as early as 4 weeks, or sometimes take 8 weeks or longer to complete . The central area of infarction may remain unhealed, showing mummified myocytes for extended periods, despite the fact that the infarct borders are completely healed. For this reason, it is important to evaluate the age of the infarct by examining the border with noninfarcted muscle.

The magnitude of repair and healing is dependent not only on infarct size but also on local and systemic factors. If there is good collateral blood flow locally, healing will be relatively rapid, especially at the lateral borders, where viable myocardium interdigitates with necrotic myocardium. There may be various levels of healing within an infarct, because of differences in blood flow in adjoining vascular beds caused by variable extents of coronary narrowing. The border areas may show hemorrhage and contraction-band necrosis, depending on regional variations in blood flow.

In humans, if reperfusion occurs within 4 to 6 h following the onset of chest pain or ECG changes; there is myocardial salvage and the infarct is likely to be subendocardial without transmural extension. There will be a nearly confluent area of hemorrhage within the infarcted myocardium, with extensive contraction-band necrosis. The extent of hemorrhage is dependent on the extent of reperfusion of the infarct as well as the extent of capillary necrosis. The larger the infarct and the longer the duration of the infarct, the greater the hemorrhage. The degree of hemorrhage may be variable and non uniform, as blood flow is dependent upon the residual area of coronary narrowing and the amount of thrombolysis. Within a few hours of reperfusion, neutrophils are evident within the area of necrosis, but they are usually sparse . In contrast to nonreperfused infarcts, neutrophils do not show concentration at the margins. However, reperfused infarcts often demonstrate areas

of necrosis at the periphery, with interdigitation with noninfarcted myocardium. Macrophages begin to appear by day 2 to 3 and stromal cells show enlarged nuclei and nucleoli by days 3 and . Neutrophil debris, which may be concentrated at the border areas in cases of incomplete reperfusion, is seen by 3 to 5 days. By days 3 to 5, fibroblasts appear, with an accelerated rate of healing as compared to nonreperfused infarcts. By 1 week, there is collagen deposition, with disappearance of neutrophils; there is also prominence of macrophages containing pigment derived from ingested myocytes. Angiogenesis is prominent and lymphocytes are often seen. Infarcts at 5 to 10 days are more cellular, and there is prominent myocytolysis (loss of myofibrils). As early as 2 to 3 weeks, subendocardial infarcts may be fully healed. Some 5 to 10 layers of subendocardial myocytes are spared without necrosis. However, myofibrillar loss, which is a result of ischemia not severe enough to cause cell death, is prominent in this subendocardial zone. Larger infarcts and those reperfused after 6 h take longer to heal. Infarcts reperfused after 6 h show larger areas of hemorrhage as compared to occlusions with more immediate reperfusion. However, myocytes maintain their striations, become stretched and elongated, and—as they do not respond to calcium influx—do not show significant contraction-band necrosis. Despite the fact that reperfusion should occur within 6 h of occlusion for maximal myocyte salvage, there appears to be some benefit in opening an artery regardless of the duration of coronary occlusion.

The No-Reflow Phenomenon

This phenomenon was first described by Kloner and Jennings in 1974.They used an experimental canine model of myocardial infarction [35] and demonstrated homogenous distribution of thioflavin S dye after 40 min of ischemia and reperfusion; however, after 90 min of ischemia, areas of no reflow were identified mainly in the subendocardial regions as zones not staining with thioflavin S. Electron microscopic examination revealed swollen endothelial protrusions and membrane-bound intraluminal bodies, which obstructed the capillary lumen and resulted in plugging of the capillaries by red cells, neutrophils, platelets, and fibrin thrombi. The areas not stained by thioflavin S were characterized by low regional myocardial blood flow.

Myocardial dysfunction associated with reperfusion of the ischemic myocardium has been termed as reperfusion injury.

TABLE-1 PATHOLOGICAL CHANGES IN MI

	PERMANENT OCCLUSION/NO REPERFUSION		REPERFUSION FOLLOWING OCCLUSION	
Time of Occlusion	**Gross**	**Histologic**	**Gross**	**Histologic**
12 h	No change/pallor	Wavy fibers	Mottled, prominent hemorrhage	CBN
24–48 h	Pallor—yellow, soft	Hypereosinophilic fibers, PMNs at borders	Prominent hemorrhage	Hypereosinophilic fibers + CBN + PMNs + hemorrhage throughout
3–5 days	Yellow center, hyperemic borders	Large number of PMNs at border, coagulation necrosis, loss of nuclei	Prominent hemorrhage	Aggressive phagocytosis profuse fibroblast infiltration + collagen77[None]
6–10 days	Yellow, depressed central infarct, tan-red margins	Mummified fibers in center, macrophage phagocytosis + granulation tissue at borders	Depressed red-brown infarct with gray-white intermingled	Aggressive healing with greater collagen
10–14 days	Gray red borders, infiltrating central tan-yellow infarct if large	Marked granulation tissue, collagen deposition, subendocardialmyocyte sparing	Gray-white intermingled with brown	Aggressive healing with greater collagen
2–8 weeks	Gelatinous to gray-white scar, greater healing at border zone	Collagen deposition with prominent large capillaries	White intermingled with groups of myocytes with red myocardium	Collagen intermingled with groups of myocy

This is assessed by contractile performance, lowering of the arrhythmogenic threshold, conversion of reversible to irreversible myocyte injury, and micro vessel dysfunction. Recent studies have shown that the angiographic no-reflow phenomenon is a strong predictor of major cardiac events, such as congestive heart failure, malignant arrhythmias, and cardiac death after MI.

GLOBAL BURDEN OF CARDIOVASCULAR DISEASES:

Cardiovascular diseases is the number one cause of deaths in all the continents of the world the solitary exception being Africa. Even in sub Saharan Africa cardiovascular diseases will be the major cause of mortality in the coming decade. According to a study the major burden of cardiovascular diseases in terms of mortality and disability occur in middle and low income countries [36]. Among all cause of deaths occurring world cardiovascular diseases alone account for around 30% of the deaths [37]. The patterns of diseases in humans have undergone major changes as a result of changes in food patterns, economy and demography. This change in the pattern of diseases is called epidemiologic transition. There is a shift from diseases related to infections to those associated with lifestyle changes[38]. This transition has occurred very rapidly in the developing countries which are unable to cope with the increasing burden.

TABLE-2 EPIDEMIOLOGICAL TRANSITION

STAGE	DESCRIPTION	TYPICAL PROPORTION OF DEATHS CAUSED BY CVD (%)	PREDOMINANT TYPES OF CVD
Pestilence and famine	Predominance of malnutrition and infectious diseases as causes of death; high rates of infant and child mortality; low mean life expectancy	<10	Rheumatic heart disease, cardiomyopathies caused by infection, malnutrition
Receding pandemics	Improvements in nutrition and public health leading to decrease in rates of deaths caused by malnutrition and infection; precipitous decline in infant and child mortality rates	10-35	Rheumatic valvular disease, hypertension, CHD, stroke
Degenerative and man-made diseases	Increased fat and caloric intake and decreased physical activity leading to emergence of hypertension and atherosclerosis; with increased life expectancy, mortality from chronic, noncommunicable diseases exceeds mortality from malnutrition and infectious diseases	35-65	CHD, stroke
Delayed degenerative diseases	CVDs and cancer are major causes of morbidity, mortality; better treatment and prevention efforts help avoid deaths in those with disease, delay primary events; age-adjusted CVD mortality declines; CVD affecting older and older individuals	40-50	CHD, stroke, congestive heart failure

The epidemiologic transition encompasses four stages pestilence and famine, receding pandemics, degenerative and man-made diseases and delayed degenerative diseases[39].In addition to this a fifth stage of obesity and inactivity

has also been described[40].Here obesity, diabetes and lipid abnormalities are increasing in children.20% of Chinese people are overweight or obese [41]

The dynamics of health transition are very different in the developing countries compared to the developed countries. Compression of the time course of the cardiovascular epidemic along with inadequate public health infrastructure coupled with low levels of education and awareness about the disease, co-existing burden of communicable diseases are all major obstacles that prevent us from taking effective measures.

In India alone around 32 million people are believed to suffering from cardiovascular diseases [42]. Approximately 2.03 million deaths have been believed to have occurred due to cardiovascular diseases by 2010. In the 30 year period from 1990 to 2020, 111% increase in deaths due to cardiovascular diseases is expected[42]. Some studies indicate women are more likely to have cardiovascular diseases than men in India[43]. More than half of the Cardiovascular deaths in India occurred in people less than 70 years.

A study conducted in south India in rural Andhra Pradesh has shown that prevalence may well be higher in rural areas[44]. Developing countries are affected a decade or two earlier than developed countries, often resulting in loss of a sole

bread- winnerof the family imposing a significant burden on the family as well as the community.

RISK FACTORS:

SMOKING:

Smoking is the single most important risk factor for coronary artery disease. Cigarette consumption is the most important cause of preventable death in the world [45]. The use of tobacco is rising among women, adolescents and young adults[46] . Around 930,000 adult deaths have been estimated to have occurred in India due to smoking related causes [47]. Smoking causes acceleration of atherosclerosis, impairs vasodilatory capacity of coronary arteries, increases levels of stress hormones, increases bronchial secretions, impairs mucociliary clearance and aggravates associated pulmonary dysfunction. Passive smoking is equally hazardous. Smoking also causes :

1.increased levels of oxidized LDL

2.lowers levels of cardio protective HDL

3.nicotine and carbon monoxide in smoke produce endothelial damage

4.increases vascular reactivity

5.lowers threshold for myocardial ischemia; increases coronary vasospasm

6.increases fibrinogen levels and platelet aggregability.

Cessation of cigarette consumption remains the single most important intervention in preventive cardiology. A study demonstrated, smoking cessation reduced cardiovascular mortality by 36% as compared with mortality in subjects who continued smoking, an effect that did not vary by age, sex, or country of origin[48].

Reductions in smoking from any mechanism improve health outcomes, especiallywhen coupled along with lifestyle changes, including exercise and dietary control. Trials of nicotine replacement therapy using transdermal nicotine or nicotine chewing gum increase abstention rates after cessation. Such pharmacologic programs, as well as physician-guided counseling, are cost-effective and should be provided as standard prevention services. Low-yield cigarettes do not appear to reduce the risks of myocardial infarction. Although the elevated cardiovascular risks associated with smoking decrease significantly after cessation, the risks of cancer of the lungs, pancreas, and stomach persist for more than a decade, as do the risks of developing chronic obstructive pulmonary disease. Smoking cessation has clear benefit, but smoking reduction alone appears to have only a marginal effect. Patients often do not understand the importance of smoking cessation. This is common especially in the developing world where low levels of literacy and awareness hamper smoking cessation programs.

Studies of social networking indicate that smoking cessation by a spouse decreases a person's chance of smoking by 67%.Smoking cessation by a brother or sister, friend, or neighbour, people who work in the same environment decreases a person's chance of smoking by 25% to 36%. Smoking cessation programs that have provided direct financial benefits to those who have stopped smoking have found to be effective. But the most important component of any effective smoking reduction strategy is health education to the community and physician based primary prevention program[49].

HYPERTENSION:

The prevalence of hypertension is steadily increasing in the Indian population. The total number of adults with hypertension in the world is anticipated to exceed 1.5 billion[50].Worldwide, hypertension causes 7.6 million premature deaths annually, with 80% of this burden occurring in low- and middle-income countries[51].

Isolated systolic hypertension, in particular, has at least as much importance as diastolic blood pressure for the outcomes of total cardiovascular mortality and stroke. There is strong evidence favouring the treatment of systolic hypertension even in the older adults .Pulse pressure, generally reflecting vascular wall stiffness, also predicts first and recurrent myocardial infarction. Pulse pressure is defined as

the difference between systolic and diastolic blood pressures. Pulse pressure appears to predict cardiovascular events independently, particularly heart failure. Home-based monitoring of blood pressure is a better indicator for vascular events than office-based evaluation [52].Nocturnal hypertension diagnosed by continuous ambulatory blood pressure monitoring is associated with an increased risk of congestive cardiac failure .Self-measurement helped in the detection of those with white coat hypertension but did not improve overall management.It also did not alter the objective measures of compliance, like left ventricular mass. Blood pressure reductions as miniscule as 4 to 5 mm Hg result in large and clinically significant reductions in risk for stroke, vascular mortality, congestive heart failure, and total CHD in people of all age groups irrespective of race such including those with diabetes and peripheral arterial disease.

Though non pharmacological measures like diet and life-style management have an important role in control of hypertension when used alone the results have been disappointing. But Diet and lifestyle changes have an important role in the prevention of hypertension and they can reduce the burden of high blood pressure in the community. Multiple drug regimens involving anti hypertensives at low doses capable of reducing systolic blood pressure by 20mm Hg and diastolic blood pressure by 11mm Hg have reduced the risk of stroke and CHD by 63% and 46% respectively[53]. The ACCORD trial(ACTION TO CONTROL

CARDIOVASCULAR RISK IN DIABETES) showed that in diabetic patients aiming for systolic BP less than 120mm Hg as compared to 140mm Hg did not reduce the rate of cardiovascular events[54]. In order to maintain clinical benefits blood pressure control must be maintained life-long. Another important aspect is that hypertension co exists with glucose intolerance, insulin resistance, hyperinsulinemia ,obesity, left ventricular hypertrophy and renal failure making its treatment complicated.

Hypertension increases risk of coronary events by

1.impairing function of the endothelium

2.increased endothelial permeability to lipoproteins

3.increased oxidative stress

4.increased adherence to leukocytes

5.acute plaque rupture due to haemodynamic stress

6.increasing oxygen demand of myocardium

7.decreasing myocardial compliance and impaired myocardial relaxation

8.increasing myocardial wall stress.

DYSLIPIDEMIA:

Cross-sectional population studies have consistently revealed a relationship between serum cholesterol levels and CHD death. The validity of such studies are however limited by the presence of multiple confounding factors. The emergence of data from prospective cohort studies, such as that begun in Framingham in the 1950s, bolstered the relationship between cholesterol and CHD. This study, as well as others performed with different populations around the world, established the concept of cholesterol more firmly as a culprit in CHD.

Although based on experimental and clinical observation, doubts persisted regarding the role of cholesterol in atherosclerosis until very recently. Through the beginning of the 1990s, controversy enveloped the role of cholesterol-lowering therapy in CHD risk reduction. Despite evidence that high cholesterol levels correlated with coronary death, the proposition that cholesterol-lowering therapy could reduce CHD morbidity remained unproven. Critics pointed to the J-shaped curve, which apparently described the relationship of serum cholesterol to mortality. Advocates of the cholesterol hypothesis countered that the heightened risk for all-cause death in individuals with low levels of cholesterol might reflect co morbidities such as cancer, inanition, or liver disease. The goal of reducing CHD mortality by drug therapy eluded convincing proof for decades. Some

cholesterol-lowering medications appeared to cause an increase in the incidence of some events, including non coronary death. In the pioneering coronary drug project, estrogen treatment led to excess mortality in the cohort of men studied. The World Health Organization study of clofibrate showed excess non coronary death. Dietary interventions to lower cholesterol often proved ineffective and, together, such results seemed to challenge the validity of cholesterol as a therapeutic target.

Conclusive evidence regarding the cholesterol hypothesis awaited clinical trials of cholesterol lowering using the hydroxyl methyl glutaryl coenzyme A (HMG-CoA) reductase inhibitors (statins), agents that lowered LDL cholesterol much more efficiently than previously available drugs. Unassailable clinical trial evidence now shows that lowering LDL cholesterol levels reduces coronary events in broad segments of the population. The ensemble of large clinical trials of statins has substantiated a decrease in total mortality in almost all major study populations. As a group, placebo-controlled trials of statins lowered LDL cholesterol levels by 20% to 60% and reduced coronary events by up to one third over a 5-year period, with no evidence of an increase in nonvascular mortality.

The Heart Protection Study showed that statins can reduce stroke and coronary events in those with preexisting vascular disease. Several head to head trials comparing different statin regimens have shown that even more aggressive LDL reduction is associated with greater clinical improvements .

The case for LDL cholesterol as a CHD risk factor thus meets most of the criteria established by Robert Koch in the 19th century to establish a causative agent in a disease. High cholesterol levels consistently predict risk of future cardiovascular events in human populations. Animal studies in multiple species have shown a causal relationship between hypercholesterolemia and atherosclerosis. Knowledge of the LDL receptor pathway plus emerging understanding of the vascular biology of atherosclerosis provides biologic plausibility for the involvement of LDL in atherogenesis. The human mutations in the LDL receptor produce hypercholesterolemia on a monogenic basis that causes accelerated atherosclerosis as early as the first decade of life in individuals with homozygous familial hypercholesterolemia. Finally, interventions in large clinical trials to lower LDL cholesterol levels by various approaches (e.g., bile acid–binding resins, intestinal bypass surgery, statins) have shown a reduction in cardiovascular events. Thus, LDL cholesterol fulfills the criteria of modified Koch's postulates as one causative agent in atherosclerosis.

Several independent lines of evidence have suggested that what is regarded as "normal" cholesterol levels in Western society exceed levels that good health requires.[55] In particular, certain rural agrarian societies with very low rates of atherothrombosis have total cholesterol levels well below those accepted as normal in Western societies. Another line of evidence derives from phylogeny. Contemporary humans have much higher total cholesterol levels than those of many other species of higher organisms that thrive nonetheless. Clinical trials have shown that LDL cholesterol levels as low as 50 mg/dL provide optimal outcomes, even in primary prevention.

Cholesterol levels measured early in life influence long-term cardiovascular risk. Substantial evidence suggests that the burden of risk for cardiovascular disease begins in young adulthood. Autopsy studies from the Korean and Vietnam conflicts, and recent explorations of coronary anatomy by intravascular ultrasonography, indicate that atherosclerosis affects adolescents in our society. In very high-risk children, clinical trial evidence has demonstrated the efficacy of statin therapy for lipid-lowering in familial hypercholesterolemia. Statin trials have now demonstrated convincing benefits on hard clinical endpoints, even among those without established cardiovascular disease. Not all patient subgroups, however, have shown such clear benefits with statin therapy. In those with severe congestive heart failure or on hemodialysis, recent trials have not found lowered

event rates in statin-treated patients, suggesting that the use of these agents late in the disease process may yield less benefit.

As is the case with LDL cholesterol, abundant prospective cohort studies have demonstrated a strong inverse relationship between high-density lipoprotein (HDL) cholesterol and vascular risk. In general, each increase of HDL cholesterol by 1 mg/dl is associated with a 2% to 3% decrease in risk of total cardiovascular disease. Patients with angiographically proven coronary artery disease more often have low levels of HDL than high levels of LDL, as defined by current criteria. The process of reverse cholesterol transport may explain in part the apparent protective role of HDL against coronary death. According to this concept, HDL could ferry cholesterol from the vessel wall, augmenting peripheral catabolism of cholesterol . HDL can also carry antioxidant enzymes that may reduce the levels of oxidized phospholipids in atheromatous lesions, which could enhance atherogenesis. Furthermore, overexpressing the apolipoprotein A-I gene in transgenic mice and infusing complexes of apolipoprotein A-I and phospholipids into hyperlipidemic rabbits not only increases HDL cholesterol levels (HDL-C) but also decreases atherosclerotic development. Evidence is also accruing of anti-inflammatory properties for HDL-C.

In contrast to LDL, however, we currently lack evidence that increasing HDL-C levels reduces risk. The Investigation of Lipid Level Management to Understand Its Impact in Atherosclerotic Events (ILLUMINATE) trial, in which the cholesteryl ester transfer protein inhibitor torcetrapib was given to patients at high vascular risk, showed an unanticipated increase in all-cause mortality,[56] findings that have led to considerable controversy as to whether HDL-C remains a viable therapeutic target.

Nonetheless, the consistency of the observational data, both cross-sectional and prospective, strongly supports the HDL level as a negative risk factor, as incorporated in the Adult Treatment Panel (ATP-III) guidelines, and supports the continued careful evaluation of agents that can directly increase HDL levels. This recognition by the ATP-III—that a biomarker can be useful without fulfilling Koch's postulates—has importance for clinical practice and has implications for several other emerging risk factors (see later). Optimism that HDL-C remains in the causal pathway for atherosclerotic events also comes from recent genetic studies evaluating the relationships of polymorphisms in HDL-related loci as predictors of incident vascular events.

Several investigators have suggested that the measurement of apolipoproteins A-I and B100 would predict cardiovascular risk better

than HDL and LDL cholesterol in clinical practice. Two recent prospective cohort studies have shown this to be the case for men[57] and women.[58] However, both of these studies also found that non-HDL cholesterol (defined as total cholesterol minus HDL cholesterol) provided clinical risk information at least as strong as that of apolipoprotein B100; this was an unsurprising observation, because non–HDL-C correlates very closely with apolipoprotein B100 levels. Furthermore, these studies found that the total cholesterol–to–HDL-C ratio remained a very strong predictor of risk, superior even to the ratio of apolipoprotein B100 to apolipoprotein A-I. Thus, despite evidence favoring apolipoproteins A-I and B100 in univariate analyses as replacements for HDL and LDL cholesterol, there remain little clinical data that use of these measures improves overall risk prediction compared with standard lipid testing.

Beyond standard chemical measures of total, LDL, and HDL cholesterol, which appropriately form the basis of current lipid screening and reduction guidelines, the amount of cholesterol carried by different classes of lipoprotein particles may influence function and vary widely among individuals. Therefore, measures of core lipid composition and

lipoprotein particle size have been hypothesized to provide better measures for risk prediction. Several lines of evidence have indicated that small LDL particles may be more atherogenic than large particles and contribute particularly to the dyslipidemia of diabetes. Currently, a number of technologies are available for the evaluation of LDL subclasses and particle size. Studies using density gradient ultracentrifugation and gradient gel electrophoresis have generally found that lipoprotein subclass identifies individuals at higher risk for coronary disease and have successfully shown a preferential benefit of lipid-lowering therapy for patients with small, dense LDL particles compared with large LDL particles. LDL particle concentration, as measured by nuclear magnetic resonance (NMR) studies, correlates well with coronary arterial lumen diameter after statin therapy and may offer predictive value for future vascular events. In both the Women's Health Study and the Multi-Ethnic Study of Atherosclerosis, LDL particle concentration measured by NMR predicted incident vascular events better than standard chemical measurement of LDL cholesterol.[41,42] However, in these studies, lipoprotein profiles evaluated by NMR were not superior to those of other standard measures, such as the total cholesterol–to-HDL-C ratio or non–HDL-C. Thus, although intriguing

pathophysiologic information regarding lipid reduction with statins and gemfibrozil has come from NMR studies, as well as density gradient studies, it remains unclear whether these novel methods of lipid evaluation add importantly to standard lipid screening in routine practice or should remain specialized tools for research and lipid clinics.

Triglyceride-Rich Lipoproteins and Cardiovascular Risk

In contrast to compelling evidence favoring a causal role for LDL-C in atherogenesis, the role of triglycerides remains controversial. Part of this controversy reflects the inverse correlation of triglyceride levels with HDL-C. Adjustment for HDL-C attenuates the relationship between triglycerides and cardiovascular disease. A recent meta-analysis has suggested that the adjusted risk ratio for coronary disease among those in the top third of triglyceride levels, compared with the bottom third, decreases from approximately 2.0 to 1.5 after accounting for HDL-C.[45] A second controversy reflects the continued recommendation in guidelines that triglycerides be measured in the fasting state, whereas much of the prognostic value of plasma triglyceride levels depends on postprandial levels. Two major cohort studies have recently reported that nonfasting triglycerides predict vascular events, independent of traditional risk

factors, but that fasting triglyceride levels show little independent relationship.[46,47]On this basis, prediction of vascular risk may need to be based on an oral triglyceride tolerance test, analogous to a glucose tolerance test used to diagnose diabetes.[48]Nonfasting triglycerides have also recently been found to predict incident stroke, again in contrast to fasting levels.[49]

For these reasons, among others, current guidelines do not establish a target value of triglycerides. However, in view of the tight link of triglyceride levels with known risk factors for atherosclerosis (e.g., low HDL-C level, uncontrolled diabetes, hypothyroidism), the finding of marked and persistently elevated triglyceride levels should enter into overall risk assessment for an individual and stimulate consideration of the reason for triglyceride level elevation, including careful exclusion of secondary causes . A cautious approach to triglyceride reduction seems prudent, because randomized trials using fenofibrate in diabetic patients have not shown significant reductions in risk when added to statin therapy.

DIABETES:

The demon of diabetes is taking a meteoric toll on the India population. India and China house a very large number of world's diabetic population. China is home to around 90 million diabetics and India the number hovers around 61 million according to a study by International Diabetes Federation (IDF) in 2011.Both

countries are in a neck and neck race to earn the dubious distinction of ‘Diabetic Capital’ of the world.

Indians in particular are affected by diabetes at a younger age, they tend to be leaner, exibhit higher degrees of insulin resistance and have abdominal obesity. According to a study, if a patient develops diabetes, the disease conferred a risk equivalent to 15 years aging [59]. This is much higher than that conferred by smoking. Diabetes causes acceleration of atherosclerosis in the major arteries in addition to increasing the atherosclerotic burden in the microvasular tone. Even before diabetes becomes evident clinically, cardiovascular risk seems to increase[54]. Insulin resistance commonly a twin of obesity, by itself increases the rate of atherosclerosis. It also increases the risk of heart failure. Diabetes leads to widespread endothelial dysfunction, increased oxidative stress, impaired fibrinolysis coupled with increased platelet aggregability. This is done through various mechanisms. Alterations in the coagulation pathway due to increased levels of tissue thromboplastin, factor vii, increased amounts of von Willebrand factor coupled with decreased levels of antithrombin iii, proteinC, proteinS, etc leads to a prothrombotic state.The accumulation of advanced glycation end products also contributes to inflammation.

TABLE -3 MECHANISMS OF DIABETIC VASCULAR DISEASE

Endothelium	↑ NF-κβ activation ↓ Nitric oxide production ↓ Prostacyclin bioavailability ↑ Endothelin 1 activity ↑ Angiotensin II activity ↑ Cyclooxygenase 2 activity ↑ Thromboxane A_2 activity ↑ Reactive oxygen species ↑ Lipid peroxidation products ↓ Endothelium-dependent relaxation ↑ RAGE expression
Vascular smooth muscle cells and vascular matrix	↑ Proliferation and migration into intima ↑ Increased matrix degradation Altered matrix components
Inflammation	↑ IL-1β, IL-6, CD36, MCP-1 ↑ ICAMs, VCAMs, and selectins ↑ Activity of protein kinase C ↑ AGEs and AGE/RAGE interactions

The insulin resistance syndrome or metabolic syndrome comprises abdominal obesity as seen by increased waist circumference ,elevated triglycerides, low HDL, elevated blood pressure, increased blood sugar values. Its definition and diagnostic criteria have varied depending on study groups and national guidelines and is mired in ambiguities. But what is not in doubt is that the metabolic syndrome leads to increased cardiovascular and all cause mortality[60]. Though central obesity ,which is calculated by measuring waist circumference,is an integral component of metabolic syndrome, there are not many studies which have measured visceral fat deposition. Visceral fat deposition is believed to be the main driver of dysmetabolism[61]. Nowadays the definition of metabolic syndrome includes a pro inflammatory state as evidenced by elevated hs CRP levels in these patients[62].

EXERCISE:

Regular exercise reduces the risk of cardiovascular diseases. Earlier it was believed for exercise to be beneficial, it must be vigorous. But recent evidence indicates that exercising in moderation confers significant cardiovascular benefits. Exercise improves insulin sensitivity, lowers LDL and total cholesterol levels, improves HDL levels and lowers triglyceride levels. These benefits tend to occur even in the absence of clinically significant weight loss.

Studies have shown that if children increase the time spent in physical activity by more than one hour daily occurrence of cardiovascular risk factors at an early age can be prevented[63].Exercise further improves insulin sensitivity and glycemic control, with major benefits for diabetic patients, including reductions in glycated hemoglobin and reduced requirements for therapy. Various studies have shown that even moderate intensity exercise has significantly reduced the incidence of diabetes. Regular exercise lowers CRP levels, improves coronary endothelial function, and appears to benefit hemostatic variables, including tissue-type plasminogen activator, fibrinogen, von Willebrand factor, fibrin, D-dimer and plasma viscosity.

Chronic exercise training appears to have a considerably favorable influence on endothelial function in the peripheral arteries and the coronary arteries. This effect is due to the increased production of vasodilating nitric oxide which leads to a decrease in oxidative stress. Regular exercise also tends to increase levels of endothelial progenitor cells. Such improvements have been linked to a reduction in adverse cardiovascular events. Finally, exercise training appears to modulate the balance between sympathetic and parasympathetic tone favorably, an effect associated with improvements in survival.

NUTRITION:

'A man is what he eats'. This old saying assumes significance in the current era because of the unhealthy food habits we tend to follow. Increased consumption of dietary salt and foods rich in trans-fatty acids coupled with lower intake of omega-3 fatty acids, fruits, and vegetables, are some of the major modifiable causes of both total deaths and deaths from cardiovascular disease (CVD). In many countries including India, the bulging waistline of the population and the associated increase in obesity, DM, and CVD are primarily due to rapid social and environmental changes transmitted mainly by dietary changes and other lifestyle changes.

Knowledge of how diet affects CVD has rapidly progressed in recent years. Earlier, the association between food and cardiovascular diseases was based primarily on data from ecologic studies and short-term experiments. But now evidence is being derived from well conducted randomized control trials and prospective cohort studies of disease endpoints (e.g., myocardial infarction [MI], CHD death) that provide direct evidence for total causal effects in addition to well-conducted trials of multiple risk markers and pathways . Conclusions are most robust when studies across these different designs provide concordant findings, with supporting evidence from in vitro and animal work, retrospective studies, and

ecologic studies. Dietary factors exert acute and chronic effects on a complex set of established and novel risk factors, mechanistic pathways, and disease conditions.

Total carbohydrate quantity consumed does not associate strongly with CHD risk, but the types and quantity of carbohydrate consumed are important determinants of health effects. Dietary fiber is comprised of nondigestible polysaccharides, resistant starch and oligosaccharides, and lignins in plants. Trials have demonstrated consistent benefits of dietary fiber on multiple CVD risk factors, including serum TG, low-density lipoprotein cholesterol (LDL-C), blood glucose, and BP. In hypertensive patients, for example, higher fiber intake reduces systolic (S) BP and diastolic (D) BP by 6.0 and 4.2 mm Hg, respectively. Unfortunately, few long-term trials have been performed. In the Diet and Reinfarction Trial in men with prior MI, advice to consume cereal fiber had no significant effect on CHD endpoints, but follow-up was limited to 2 years. In contrast, in long-term prospective cohorts, fiber from grains, cereals, and fruits is associated with a lower incidence of CHD, and fiber from grains and cereals with a lower incidence of DM. Cereal fiber intake may also reduce risk via a substitution effect, replacing more refined carbohydrates that may have detrimental effects.

Types of Fat

In contrast to the relatively limited health effects of the proportion of energy consumed from total fat, substantial health effects can occur from increases or decreases in specific types of fats consumed, either as a replacement for other fats or for carbohydrates. Nomenclature schemes and dietary recommendations for fats traditionally follow broad chemical classifications defined by the degree of unsaturation (e.g., saturated, monounsaturated, polyunsaturated) or the type of double bond (e.g., omega[n]-3 or omega[n]-6; . Such broad groupings obscure substantial differences in the dietary sources and biologic effects of individual fatty acids within each class that can have very specific effects on gene transcription, cell membrane fluidity and function, and metabolites generated. As these distinct biologic properties are elucidated, clinical and public health focus should focus on the types and amounts of individual fatty acids. This chapter follows the conventional groupings, but also highlights some effects of individual fatty acids.

Saturated Fatty Acids

Meats, dairy products, and tropical oils (e.g., palm, coconut) are major sources of saturated fatty acids (SFAs). Based on ecologic comparisons, effects on LDL-C, and animal experiments, SFA intake would be expected to increase CHD risk. When replacing equivalent calories from carbohydrates, for example, each 1%E

greater intake of SFA increases LDL-C by 0.032 mmol/liter. But recent evidence seems to suggest otherwise. Lauric acid (12:0), myristic acid (14:0), and palmitic acid (16:0) raise the levels of LDL-C , but stearic acid (18:0) does not seem to have this effect. An important thing to note is that since dietary factors affect chronic disease via multiple pathways, effects on any single risk marker cannot be assumed to translate directly into changes in disease incidence. For example, the 12-, 14-, and 16-carbon SFAs raise LDL-C compared with carbohydrates, but they also lower TG, raise HDL-C, and raise apolipoprotein A-I (apo A-I) levels.SFAs also lower lipoprotein(a) when consumed in place of monounsaturated fatty acids (MUFAs) or carbohydrates. In the setting of multiple complex lipid and lipoprotein changes, effects on a more global lipid risk marker may be most informative. These changes suggest minimal effects or even small CHD benefits of SFAs compared with carbohydrates.

Faced with conflicting evidence from risk markers, prospective cohorts and trials of disease endpoints may provide better evidence for total clinical effects. In the large WHI trial, SFA intake was reduced from approximately 12.5% to 9%E—largely replaced with carbohydrates—without effects on incident CHD (RR, 0.98), stroke (RR, 1.02), total CVD (RR, 0.98), or diabetes (RR, 0.96).[64,65] Similarly, two systematic reviews and meta-analyses of prospective cohort studies found no significant association between SFA intake and incident CHD [66].These three large

studies indicate no overall effect of SFA consumption on CHD events. Evidence from a pooled analysis of individual-level data, including 344,696 men and women in 11 prospective cohorts in the United States, Europe, and Israel, suggests that the effects of SFAs on CHD may depend on the nutrient replaced. Consuming SFAs in place of carbohydrates was associated with lower CHD risk , whereas consuming SFAs in place of polyunsaturated fatty acids (PUFAs) was associated with higher risk, consistent with some clinical trials . Observational analysis of replacing SFAs with MUFAs is limited by their common dietary sources, and no clinical trials of CHD events have tested the effects of replacing SFAs with MUFAs.

These lines of evidence, together with the favorable effects of PUFAs on CVD risk factors, suggest that PUFA intake is beneficial in place of SFA or carbohydrates (see later), but that replacing SFAs with carbohydrates may cause little benefit or even slight harm. Data on effects of carbohydrate (see above) would suggest that carbohydrate quality or individual susceptibility to insulin resistance would modify this latter effect; indeed, a recent cohort demonstrated that SFA consumption was associated with significantly lower CHD risk compared with high-GI (highly refined) carbohydrates, similar risk compared with medium-GI carbohydrates, and a trend toward higher risk compared with low-GI carbohydrates. Evidence for CVD effects of replacing SFAs with MUFAs is mixed (see later). Thus, a focus on decreasing SFAs alone may not result in substantial intended CHD

Six controlled trials have evaluated the effects of SFA versus various replacement nutrients on glucose-insulin homeostasis among individuals predisposed to insulin resistance. Three of five trials found improvements in some glucose-insulin biomarkers when SFAs were replaced with MUFAs; one of three trials, with PUFAs; one trial, with carbohydrate; and zero of two trials, with trans fatty acids (TFAs). Four controlled trials have evaluated similar questions among generally healthy individuals. Only one of four trials found improvements when SFAs were replaced with MUFAs; one of two trials, with carbohydrate; and zero of one trial, with PUFAs or TFAs. These mixed findings do not allow robust conclusions on the effects of dietary fats on glucose-insulin metabolism; the most promising evidence is for potential benefits when SFAs are replaced with MUFAs among those predisposed to insulin resistance. In observational studies, circulating or tissue SFA biomarkers are often associated with insulin resistance, but endogenous SFA levels are increased by both SFA and carbohydrate consumption and also altered by lipolysis, lipogenesis, and beta oxidation. SFA intake was lowered in the WHI trial without effects on HOMA-IR or incident DM. Among four large prospective cohorts evaluating dietary fats and incident DM, none found independent associations between SFA or MUFA intake and DM. In contrast, all four cohorts found protective associations of PUFAs, vegetable oils, and/or the

ratio of PUFA to SFA intake with incident DM. These results point to PUFAs as protective against DM, consistent with findings for CHD.

Evidence for the effects of SFAs on other CVD risk factors or endpoints, such as BP or stroke, is limited. Causal mechanisms and independence from other nutrients in animal fats and proteins require further study.

Monounsaturated Fatty Acids

Animal fats and vegetable oils (e.g., olive and canola) are each major sources of MUFAs, largely oleic acid (18:1n-9). Compared with carbohydrates, MUFA intake lowers LDL-C and TG, raises HDL-C, and lowers BP. Compared with SFAs, MUFA intake lowers LDL-C and raises lipoprotein(a), without substantial change in TG or HDL-C. Fewer studies have compared MUFAs and PUFAs; as a replacement for carbohydrates, MUFAs may raise HDL-C slightly more and lower LDL-C and TG slightly less than PUFAs, with a similar overall improved TC/HDL-C ratio. Trials testing the effects of MUFAs on glucose-insulin homeostasis have shown mixed results compared with SFA or carbohydrates. Four large prospective cohorts have found no relationship between MUFA intake and incident DM.

Relatively few individual prospective studies have reported on the relationship between MUFA consumption and CHD events, with inconsistent results. In a data analysed from 11 cohorts, higher MUFA intake, as an isocaloric replacement for SFA, was associated with increased risk of cardiovascular disease. In nonhuman primates, SFA and MUFA intake were both similarly proatherogenic. No randomized controlled trials have tested whether MUFA intake reduces CHD events compared with carbohydrates, SFAs, or PUFAs.

Animal experiments have suggested that MUFAs alter LDL-C particle composition, increasing cholesteryloleate content, a change that may increase atherogenicity. This could explain why MUFAs lower LDL-C concentrations and improve the TC/HDL-C ratio but might not reduce CHD risk. In contrast, observational studies and randomized trials have consistently shown that overall dietary patterns that have included MUFAs, typically from olive oil as part of an overall Mediterranean-type diet, improve CHD risk factors and lower CHD events. It is unclear whether these benefits derive from protective effects of MUFAs per se, other related components in olive oil (in particular polyphenols in extra virgin olive oil), or other factors in the Mediterranean-type dietary pattern.

Polyunsaturated Fatty Acids

Dietary PUFAs can be classified broadly into n-6 PUFAs, largely linoleic acid (LA; 18:2n-6) from vegetable oils, and n-3 PUFAs, including alpha-linoleic acid (ALA; 18:3n-3) from plant sources (e.g., flaxseed, canola, walnuts, soybeans), and eicosapentaenoic acid (EPA, 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-3) from fish and shellfish. LA and ALA are essential fatty acids that cannot be synthesized by humans. Humans synthesize relatively little EPA and even less DHA, so that seafood consumption provides the major source. The ratio of n-6 to n-3 fatty acids is not a useful metric of health effects compared with absolute consumption levels of these dietary fats.

Linoleic Acid

LA typically comprises more than 90% of dietary PUFAs. Compared with carbohydrates, LA lowers LDL-C and TG, raises HDL-C, and improves TC/HDL-C ratio. Effects on other CHD risk markers are less established; some trials have suggested that LA may be anti-inflammatory or improve insulin resistance, but findings have been mixed. In a pooled analysis from 11 cohorts, greater PUFA intake in place of SFAs was associated with a significantly lower incidence of CHD. PUFA intake was also associated with lower CHD risk when replacing carbohydrates. Consistent with observational studies, a meta-analysis of randomized trials that increased total PUFAs or LA in place of SFAs demonstrated

reduction in CHD events. No clinical trials have tested whether consuming PUFAs in place of carbohydrates or MUFAs reduces CHD events. Overall, the evidence suggests that total PUFA or LA intake reduces CHD risk, whether in place of SFAs or carbohydrates. Relationships between PUFA intake and DM were discussed earlier .

Alpha-Linoleic Acid

In some controlled trials, ALA intake has favorably affected some CVD risk markers related to platelet function, inflammation, endothelial function, and arterial compliance; a meta-analysis of 14 trials found improvements in fibrinogen and fasting glucose levels. Whether such effects are caused directly by ALA or by its longer-chain metabolites (e.g., EPA) is unclear. Ecologic studies have suggested benefits of increasing ALA consumption in populations with low overall n-3 PUFA intake. Prospective cohorts have shown mixed results, with overall no significant relationships seen between ALA intake and CHD events. One trial in the 1960s demonstrated no significant change in CHD events with ALA supplementation, but follow-up was limited to 1 year. No other trials have tested the effects of ALA intake on CHD events, although several trials are planned or ongoing. Because ALA is an accessible and inexpensive source of n-3 PUFAs, better understanding of its effects is essential.

Eicosapentaenoic Acid and Docosahexaenoic Acid

Controlled trials have demonstrated clear benefits of marine n-3 PUFAs on heart rate, BP, and TG levels, and potential benefits on cardiac relaxation and efficiency, inflammatory responses, endothelial function, autonomic tone, and urine proteinuria. Small trials of prevention of recurrent ventricular tachyarrhythmias in patients with implantable cardioverter-defibrillators (ICDs) have yielded inconsistent results. Meta-analyses of observational and clinical trial data have consistently indicated that longer-chain n-3 PUFAs reduce CHD events, especially fatal CHD or arrhythmic death in agreement with evidence from dog and primate models for the prevention of ischemia-induced ventricular fibrillation. Four of five large randomized controlled trials of fish or fish oil intake have demonstrated significant reductions in CHD events. In a meta-analysis of randomized trials, fish oil supplementation reduced total mortality by 17% ; these populations were generally higher risk, and effects on total mortality should be more modest in populations at lower risk of ischemia-induced arrhythmic death. Observational studies have suggested benefits for other endpoints, such as nonfatal MI, ischemic stroke, and atrial fibrillation, but intervention trials have not established these benefits. In a randomized open-label trial in 18,645 Japanese subjects with statin-treated hypercholesterolemia, the addition of EPA (1.8 g/day for 4.6 years) reduced nonfatal coronary events by 19% ($P = 0.01$). Most studies have assessed combined

intakes of EPA plus DHA; insufficient evidence exists to make recommendations about EPA versus DHA separately.

Trans Fatty Acids

TFA are unsaturated fats with at least one double bond in a trans configuration. Major dietary sources are foods made with partially hydrogenated oils, such as baked goods, deep-fried foods, packaged snacks, and shortening used for home cooking. Ruminant (e.g., cow, sheep, goat) meats and milk contain small amounts of TFAs, formed by gut microorganisms. Compared with TFAs from partially hydrogenated oils, the low levels of ruminant TFAs consumed do not appear to increase CVD risk appreciably. Higher amounts of TFA intake have clear adverse lipid effects, including raising LDL-C, TG, and lipoprotein(a), lowering HDL-C, and increasing TC/HDL-C and apo B–to–apo-A-I ratios. In contrast to other macronutrients, many of these adverse effects occur regardless of the type of nutrient replaced. In controlled feeding trials, consuming 1%E from TFAs in place of SFAs, MUFAs, or PUFAs raised the TC/HDL-C ratio by 0.031, 0.054, and 0.67, raised apo B by 3.5, 10.0, and 10.9 mg/liter, raised lipoprotein(a) by 3.8, 1.4, and 1.1 mg/liter, and decreased apo A-I by 7.0, 5.3, and 5.3 mg/liter, respectively. Based on controlled trials, observational studies, and animal experiments, TFAs may also promote inflammation, endothelial dysfunction, insulin resistance,

visceral adiposity, and arrhythmia. The strength of the evidence for these nonlipid effects varies, but the implicated pathways suggest an unusual constellation of effects on adipocyte dysfunction and insulin resistance. In prospective cohorts, small amounts of TFA intake were associated with a substantially higher risk of CHD and sudden death .Numerous TFAs exist, each with varying dietary sources and biologic and physiologic effects. Emerging evidence suggests that 18-carbon TFAs, especially trans-18:2 isomers, may be most adverse.

Dietary Cholesterol

Dietary cholesterol raises both LDL-C and HDL-C, with a net increase in total cholesterol–to–HDL-C ratio of 0.02 units/100 mg dietary intake. In animal experiments, dietary cholesterol is proatherogenic. Long-term prospective studies generally have shown no significant associations of dietary cholesterol or selected dietary sources (e.g., eggs, shellfish) with incident CVD. Conversely, higher cholesterol, egg, or shellfish consumption is associated with a higher incidence of DM in five cohorts and with higher CVD risk in patients with established DM in three cohorts, suggesting potential interactions between dietary cholesterol, DM susceptibility, and CVD that require further study.

Protein

CVD effects of dietary protein have been relatively understudied. In short-term trials, protein intake in place of carbohydrates improves BP, TG and LDL-C levels, and possibly glycemic control. In the setting of stable weight, higher protein diets lower HDL-C when replacing unsaturated fat. In some studies, plant but not animal protein sources in diet associate with lower CHD risk, suggesting that types of foods consumed or overall diet patterns may be more relevant than protein per se. Four cohorts have observed protective associations between animal protein intake and risk of hemorrhagic stroke; this emerging relationship requires further study.

Antioxidants and Vitamins

B vitamins (e.g., thiamin) are diet-derived, water-soluble, and renally excreted. B vitamin deficiency is a known cause of cardiomyopathy (beriberi) in developing countries, and emerging evidence suggests that patients with chronic heart failure may commonly have lower B vitamin levels. Whether this latter deficiency relates to poor nutritional status, diuretic-induced urinary loss, or other metabolic causes—or whether replacement improves clinical outcomes—remains unknown.

Several dietary vitamins and nutrients are associated with lower CVD risk in observational studies, but multiple trials of supplements, including folate, B

vitamins, beta-carotene, vitamin C, vitamin E, and selenium, have shown no significant effects on atherosclerosis progression or CVD events . Many of these trials, for reasons of power, evaluated individuals with established CVD or clinical risk factors, whereas most observational studies evaluated generally healthy individuals. Thus, discrepancies in findings could partly be related to different time periods of biologic sensitivity (i.e., some vitamins and nutrients could be important only early in the disease course). Such explanations should be considered speculative until confirmed in prospective studies and trials. Discrepancies between observational studies and supplement trials more likely relate to residual bias in observational studies from other lifestyle behaviors (i.e., observed benefits are not caused by diet) or because of other nutritional factors (i.e., observed benefits are caused by diet but not by the specifically identified vitamins or nutrients). Diets higher in beta-carotene and other vitamins, for example, are often rich in fruits and vegetables that contain a number of other beneficial factors, including other antioxidants, minerals, phytochemicals, and dietary fiber, as well as having replacement effects for less healthy foods. Thus, isolating one or even several components would unlikely produce similar effects as from consuming the whole food, as seen in short-term trials of fruits and vegetables versus supplements.

Two cohort studies have found that higher plasma vitamin D levels, are associated with a lower risk of CVD events. But various trials involving Vitamin D supplementation did not support this finding. Studies involving vitamin D supplementation to post menopausal women also did not confer a lower risk for cardiovascular events. A meta-analysis of 18 vitamin D trials involving 57,311 participants showed only a modest benefit with regard to total mortality. But the question whether this result was related to fewer deaths from CVD or cancers, which might be prevented by vitamin D, remained unanswered. High plasma vitamin D levels may be needed for CVD benefits; But exposure to sun for brief periods will provide the required levels of vitamin D.

Marine n-3 PUFAs are a notable exception to discordance between observational studies and supplement trials. Observational studies of habitual fish intake and controlled trials of fish oil supplements have shown similar effects on CVD risk factors; numerous prospective cohorts of generally healthy individuals have demonstrated lower risk of CHD death with fish intake; four of five randomized controlled trials in individuals with and without established CHD have demonstrated significant reductions in CVD events with fish or fish oil intake; and, in a meta-analysis of controlled trials, fish oil supplementation lowered total mortality .

Flavonoids

Flavonoids are bioactive polyphenols that include flavonols (in onions, broccoli, tea, and various fruits), flavones (in parsley, celery, and chamomile tea), flavanones (in citrus fruits), flavanols such as catechins and procyanidins (in cocoa, apples, grapes, red wine, and tea), anthocyanidins (in colored berries), and isoflavones (in soy). In laboratory experiments and short-term trials, flavonoids lower BP and exhibit antioxidant, antiplatelet, and anti-inflammatory effects. In trials, intake of cocoa or dark chocolate improves endothelial function and reduces SBP and DBP (−5.9 and −3.3 mm Hg). BP lowering occurs with as little as 6.3 g (30 kcal)/day of dark chocolate, increases over time, and is related to increased endothelial nitric oxide production. This restorative mechanism suggests important benefits beyond lowering of BP, because nitric oxide–related endothelial dysfunction is fundamental to atherosclerotic and metabolic diseases. A few short-term trials of other dietary sources (e.g., tea, red wine or grapes) or of specific flavonoid extracts have not consistently found improved endothelial function, BP, or lipid levels. In prospective cohorts evaluating total or selected dietary flavonoids and risk of CVD, 9 of 12 cohorts observed lower CHD death (95% CI, 0.71 to 0.92). Heterogeneity of specific flavonoids and their dietary sources limits inference for class effects, but observed lower risk of CHD death plus endothelial

and BP benefits of cocoa or dark chocolate provide strong impetus for further study.

TABLE- 4 RISK FACTORS AND INTERVENTIONS:

CLASS	RISK FACTOR	INTERVENTION
1	Smoking	Smoking cessation
	Dyslipidemia	Lipid management
	High blood pressure	Blood pressure management
	Preventive medications	Aspirin, angiotensin-converting enzyme inhibitor, beta blocker
2	Diabetes, prediabetes	Diabetes management
	Physical inactivity	Activity management
	Overweight, obesity	Weight management
	Unhealthy diet, alcohol	Improved diet
	Inflammation	Various interventions
3	Menopause, hormone replacement therapy	
	Micronutrients	
	Psychological factors	
	Novel biochemical and genetic markers	

MATERIALS AND METHODS

MATERIALS AND METHODS:

This study was an observational cross sectional study conducted in Tirunelveli Medical College, Department of Medicine, during the period from of August 2013 to August 2014. The aim was to study the symptomatology, the type of MI, risk factors and the outcome at the end of first week in patients with ST elevation Myocardial Infarction. The necessary clearances from the concerned departments and the ethical committee was obtained prior to the start of the study.

All patients admitted to the Intensive Coronary Care Unit in whom a diagnosis of ST elevation Myocardial Infarction was made based on clinical features, ECG changes of ST segment Elevation and elevated cardiac biomarkers were considered for the study. Patients were included in the study based on inclusion and exclusion criteria.

An oral consent was taken from all patients for a detailed clinical history and examination and the required laboratory investigations. The details collected from each patient was entered in the proforma. (Annexure-1) .

The details of the patients regarding age, sex,presentingsymptoms,risk factors like smoking, alcohol intake, food habits, diabetes, hypertension, family history of coronary artery disease were recorded. The vital signs like pulse and blood pressure were recorded and Body mass index was calculated.

All the patients were subjected to routine laboratory investigations like Renal function tests, Random blood sugar and Lipid profile. Serum Creatinine Phosphokinase MB(CPK-MB) levels were also measured. Once the patients stabilized, an Echocardiogram was taken and the patient was discharged usually at the end of the first week.

TRIGLYCERIDES :

Methodology :

Calorimetric, enzymatic method with glycerophospate oxidase is commonly done . This reagent is based on the method of wako and the modifications by McGowan et al and Fossati et al.

PRINCIPLE :

Triglycerides $+H_2O \rightarrow$ glycerol + free fatty acids

Glycerol + ATP $\rightarrow$ Glycerol 3 phosphate + ADP

Glycerol 3 phosphate + $O_2 \rightarrow$ DAP + H_2O_2

H_2O2 + 4AAP +3,5DHBS $\rightarrow$ Quinoneimine dye + $2H_2O$

The intensity of quinoneimine formed is proportional to the triglycerides concentration in the sample when measured at 505 nm(500-540nm)

TABLE -5.PROCEDURE :

Pippette in tubes marked	blank	standard	test
Working reagent	1000 ul	1000ul	1000ul
Distilled water	10ul	-	-
Standard	-	10ul	-
Sample	-	-	10ul

Mix and incubate for 10 minutes at 37 degree Celsius, read the absorbance of standard and each sample at 505/670 nm on biochromatic analysers against reagent blank.

CALCULATION :

Triglycerides (mg/dl) = abs of test/abs of standard * concentration of std (mg/dl).

CHOLESTEROL :

METHODOLOGY :

The method is based on the Trinders methodology

PRINCIPLE ;

Cholesterol ester + H_2O → Cholesterol + fatty acids

Cholesterol + O_2→ Cholest-4-en-3-one + H_2O_2

$2H_2O_2$ + 4AAP + Phenol →Quinoneimine dye + $4H_2O$

Absorbance of Quinoneimine so formed is directly proportional to cholesterol concentration.

TABLE-6.PROCEDURE :

Pippette in tubes marked	blank	Standard	test
Working reagent	1000 ul	1000ul	1000ul
Distilled water	10ul	-	-
Standard	-	10ul	-
Sample	-	-	10ul

Mix well and incubate for 5 minutes at 37 degree c or 10 minutes at 20 -25 degree c. read the absorbance of the test and standard against reagent blank.

CALCULATION :

Cholesterol (mg/dl) = abs of test/abs of standard * concentration of std (mg/dl).

HDL DIRECT :

The assay is based on a modified polyvinyl sulfonic acid and polyethylene glycol methyl ether coupled classic precipitation method with the improvements in using optimised quantities of PVS/PEGME and selected detergents. LDL,VLDL and chylomicron react with PVS and PEGME and the reaction results int eh

inaccessibility of LDL,VLDL and chylomicron by cholesterol oxidase and cholesterol esterase.The enzymes selectively react with HDL to produce H_2O_2 which is detected through a trinder reaction.

HDL + LDL+VLDL+CM → HDL +(LDL+VLDL+CM) + PVS/PEGME

HDL → fatty acid + H_2O_2

$2H_2O_2$ + 4-AA +TODB → Quinone +$5H_2O$

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2010) developed by Centre for Disease Control, Atlanta, USA .

OBSERVATIONS AND RESULTS

OBSERVATIONS AND RESULTS

200 patients admitted with clinical features and ECG changes suggestive of ST elevation Myocardial Infarction were enrolled in the study.

SEX DISTRIBUTION:

TABLE-7

Sex	Cases	
	No	%
Male	155	77.5
Female	45	22.5
Total	**200**	**200**

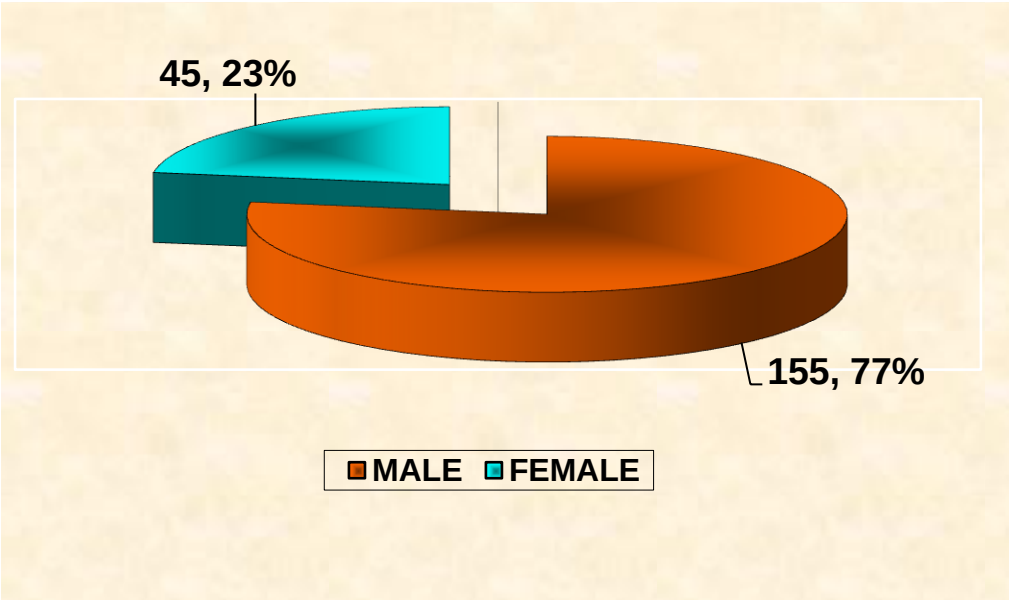

FIG.10 SEX DISTRIBUTION

AGE DISTRIBUTION:

In the study the youngest patient was a 28 year old male and the oldest patient was a 84 year old male.36 cases (18%) were younger than 45 years, the criteria for young MI. 164 cases (82%) were above 45 years.33 cases (16.5%) had an age more than 70 years. The mean age group was 57.7 years.

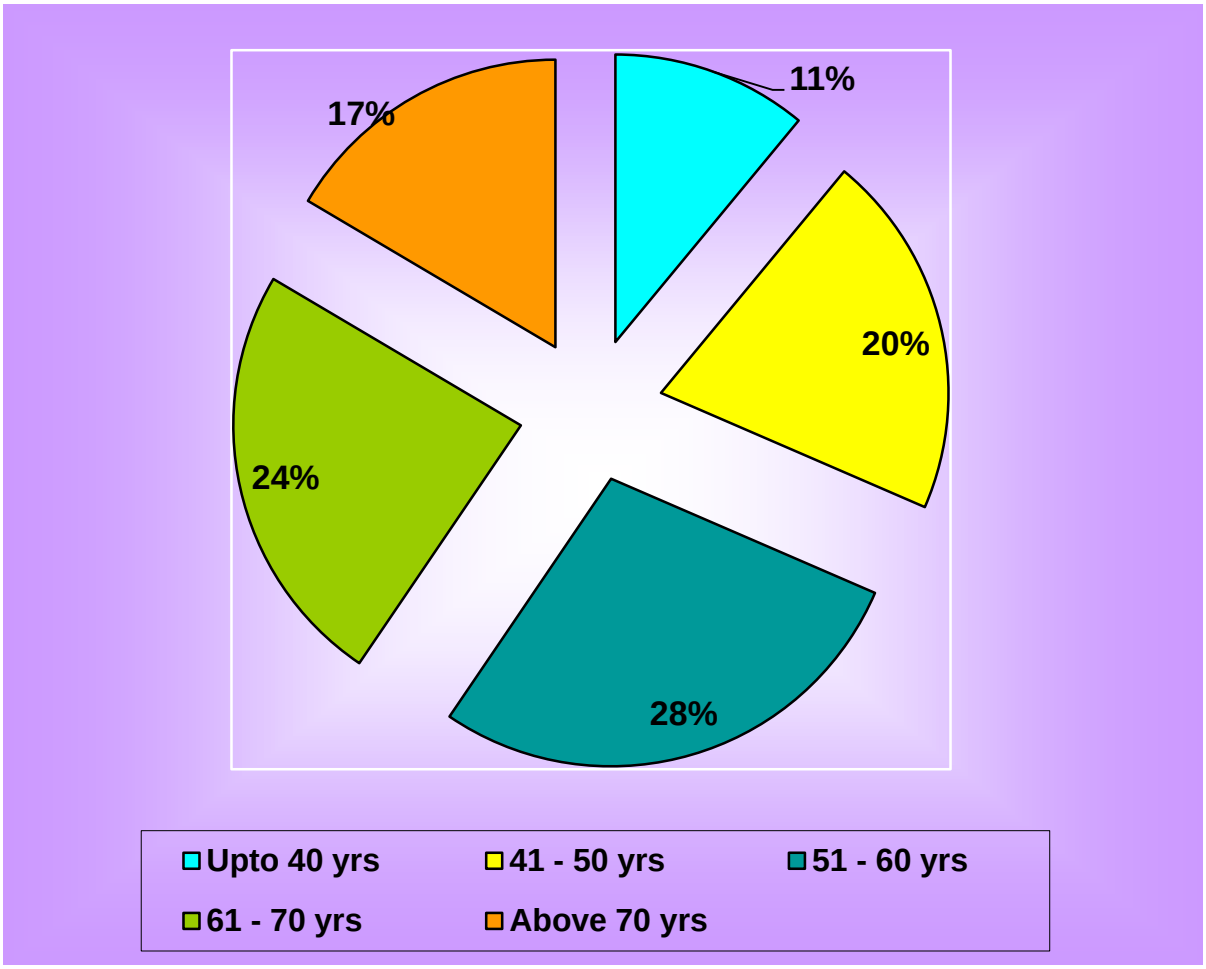

FIG.11 AGE DISTRIBUTION

TABLE-8 AGE DISTRIBUTION

Age Group	Cases	
	No	%
Upto 40 yrs	22	11.0
41 – 50 yrs	41	20.5
51 – 60 yrs	56	28.0
61 – 70 yrs	48	24.0
Above 70 yrs	33	16.5
Total	200	200
Young Group (<45)	36	18.0
Old Group (>45)	164	82.0
Range	28 – 84 yrs	
Mean	57.7 yrs	
SD	12.0 yrs	

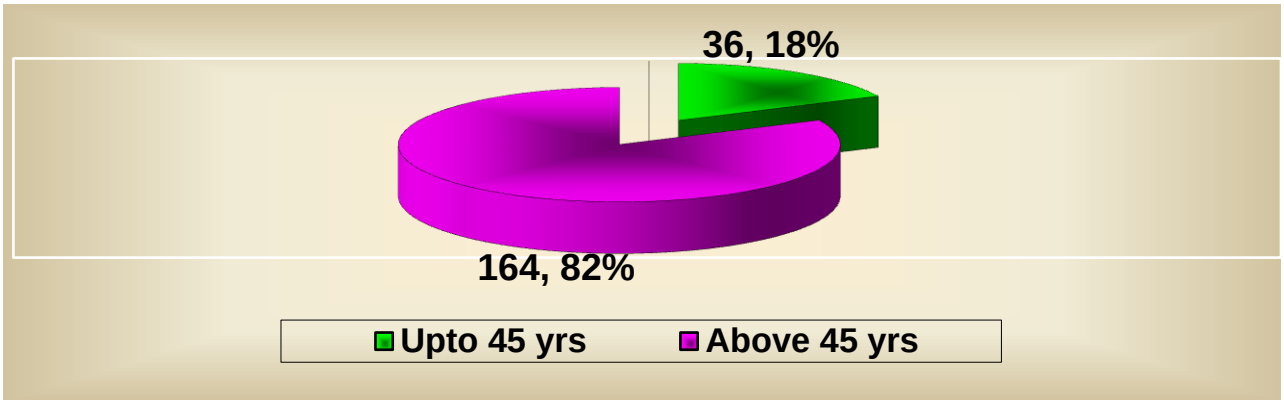

FIG.12 AGE < 45 YRS VS. AGE>45 YRS

PRESENTING COMPLAINTS:

Around 134 patients (67%) came with chest pain as the major complaint.41 cases(20.5%) came with giddiness as the main presenting feature. 25 cases came with breathlessness as the main complaint. One patient had only sweating as the main complaint. Among the 33 cases over 70 years of age, 25 cases (75.7%) had giddiness or breathlessness as the presenting feature.

TABLE-9 PRESENTING COMPLAINTS

Complaints	**Cases**	
	No	**%**
Breathlessness	25	12.5
Chest Pain	134	67.0
Sweating	1	0.5
Giddiness	41	20.5
Total	**200***	**200**

*One patient had more than one complaint

FREQUENCY OF SYMPTOMS AMONG PATIENTS:

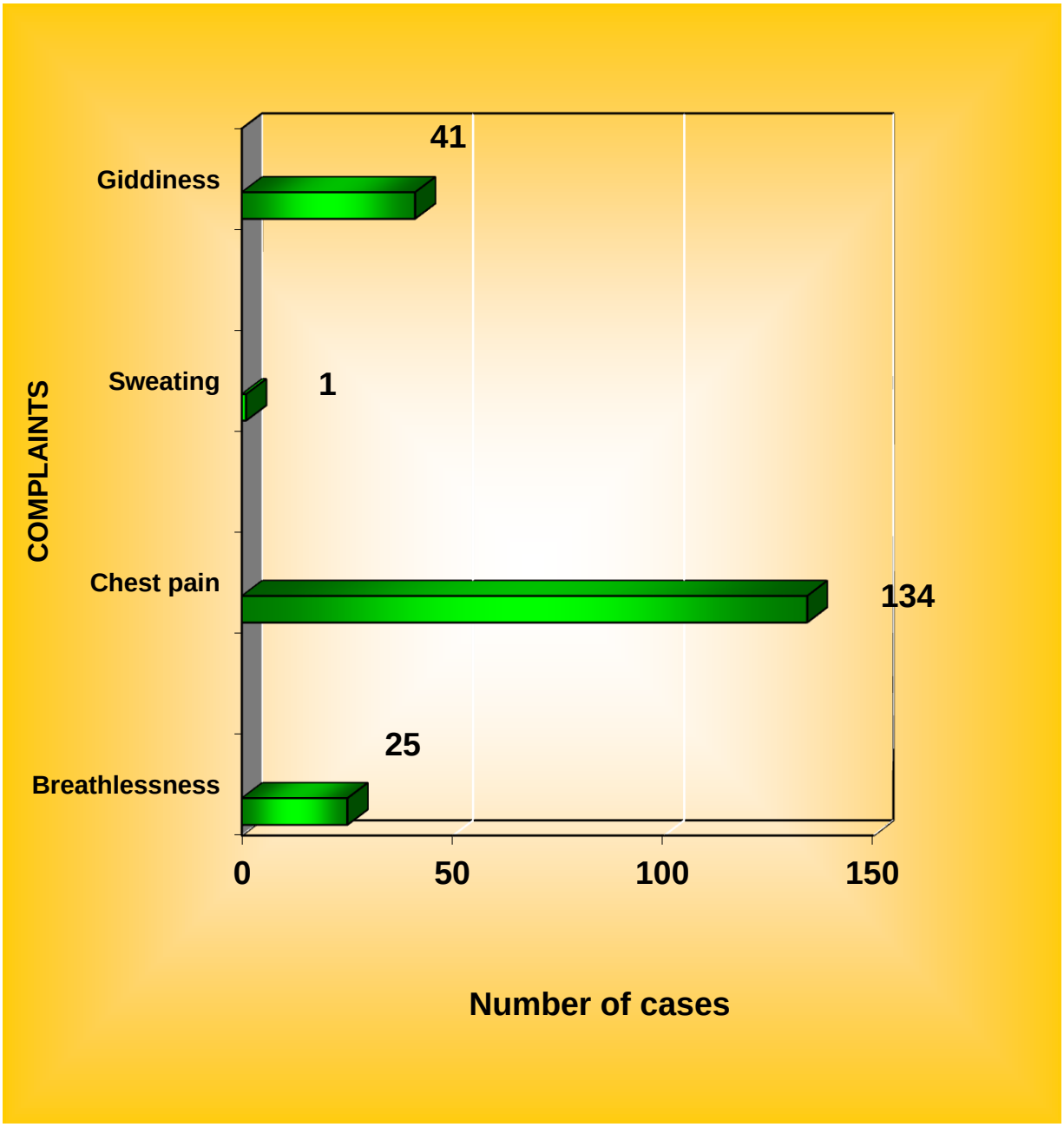

FIG.13 FREQUENCY OF SYMPTOMS

VITAL SIGNS:

The mean pulse rate of the group was around 82.2 with a lowest being 42/min and the highest being 116/min. The mean Systolic BP was 140.4 mm Hg with a range between 80 mm Hg and 190 mm Hg. The mean diastolic BP was 86.5 mm Hg with a range between 50 mm Hg and 130mm Hg.

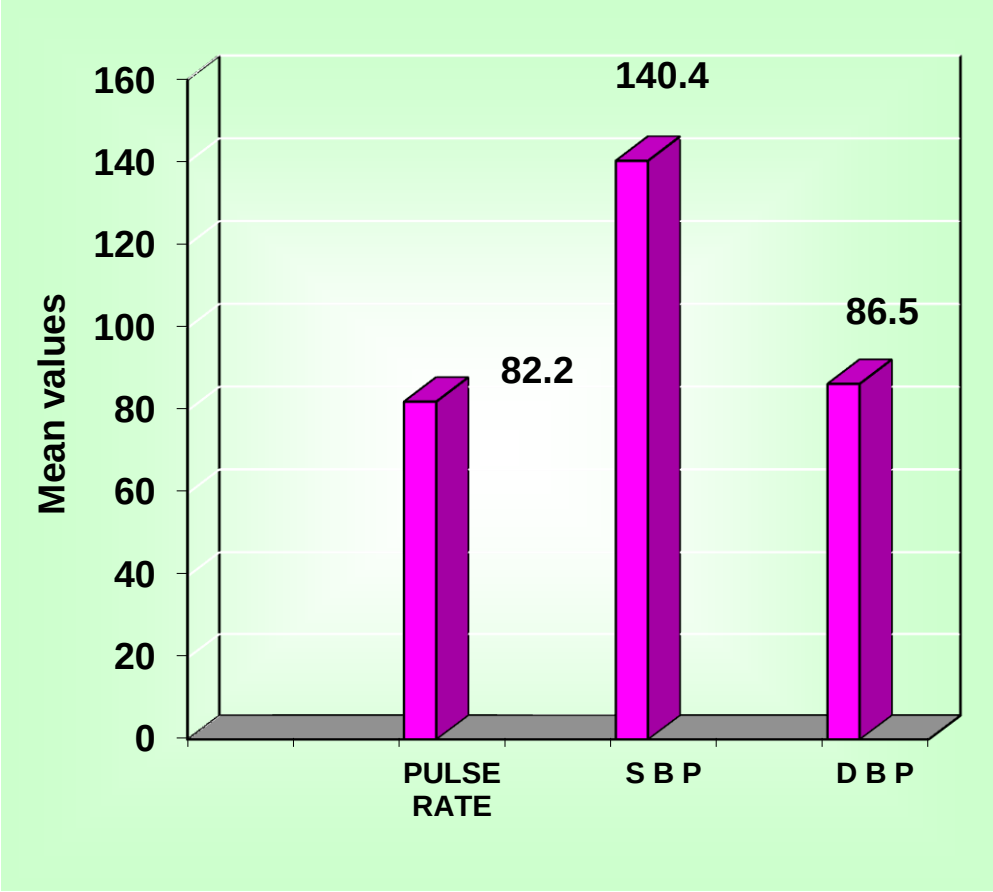

FIG.14 VITAL SIGNS

TABLE-10 VITAL SIGNS

Variable	Range	Mean	SD
Pulse Rate	42 - 116	82.2	16.0
Systolic BP	80 - 190	140.4	28.5
Diastolic BP	50 - 130	86.5	15.8

FOOD HABITS:

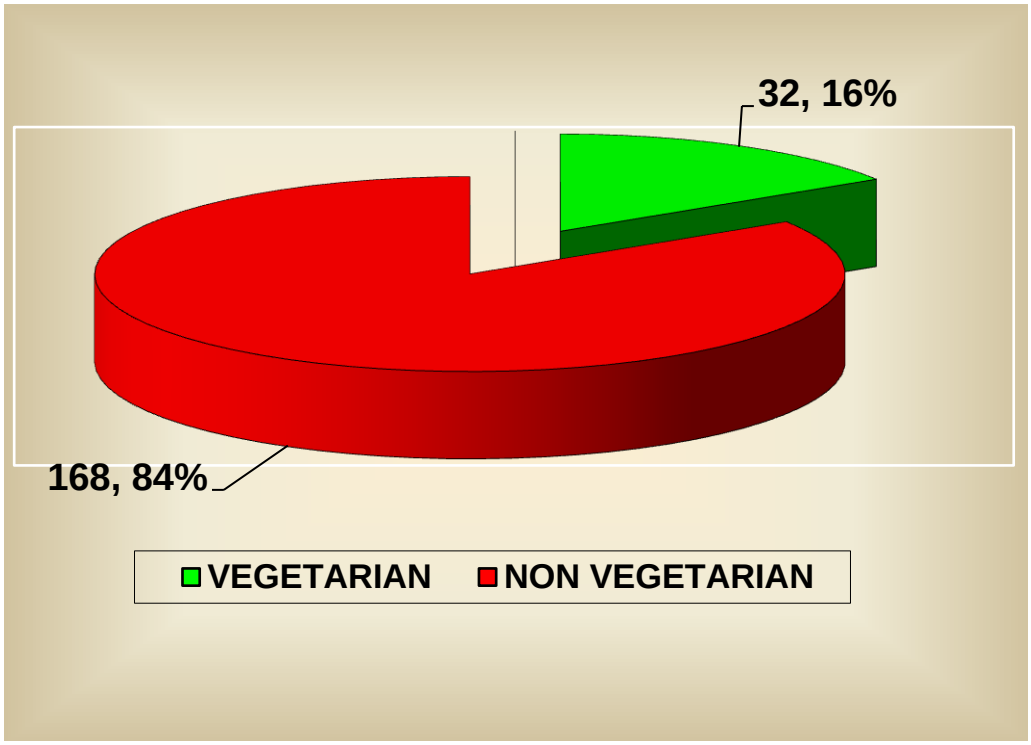

FIG.15 FOOD HABITS

TABLE-11 FOOD HABITS

Food Habit	Cases	
	No	%
Vegetarian	32	16.0
Non Vegetarian	168	84.0
Total	200	200

32 cases (16%) of the study group were vegetarian; the remaining 168 cases (84%) being non vegetarian.

BODY MASS INDEX:

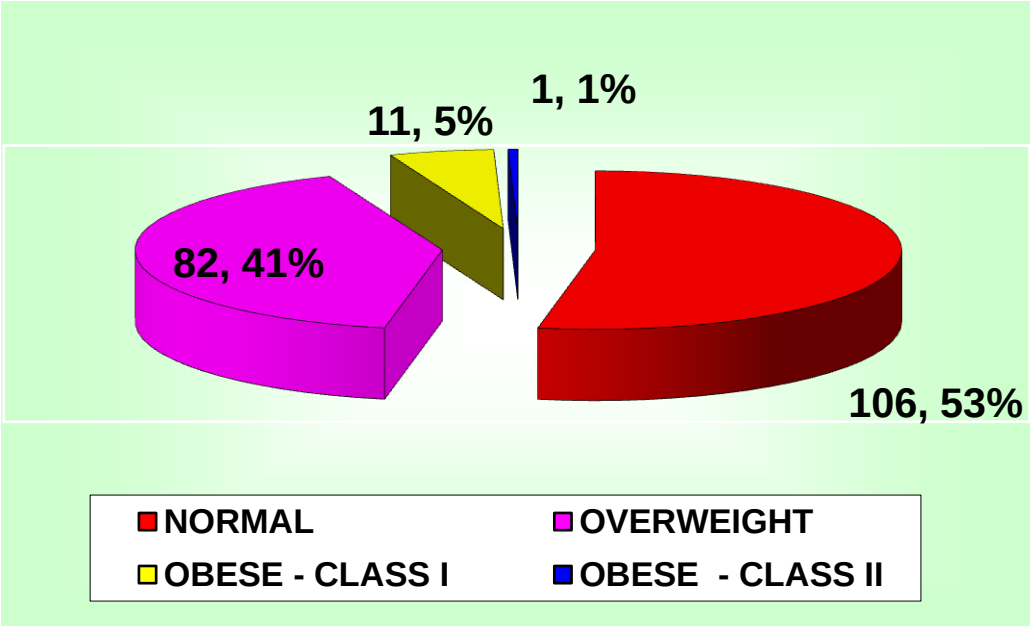

FIG.16 BODY MASS INDEX

TABLE-12 BODY MASS INDEX OF PATIENTS

BMI	**Cases**	
	No	**%**
Normal (18 – 25)	106	53.0
Over weight (25 – 30)	82	41.0
Obese Class I (30 – 35)	11	5.5
Obese Class II (35 – 40)	1	0.5
Total	**200**	**200**
Range	18.3 – 35.15	
Mean	25.57	
SD	3.13	

106 cases (53%) had a normal BMI with 82 (41%) of them being in the overweight category.12 cases (6%) belonged to the obese category. The mean body mass index is around 25.6 with a range from 18.3 to 35.15

BLOOD SUGAR/UREA/CPK-MB LEVELS:

The average blood sugar value was 152.7 mg% with a range between 60 mg %and 321 mg%. The mean urea values were 35.9 mg% and CPK-MB was 36.7 IU/L

TABLE-13 SUGAR /UREA/CPK-MB OF PATIENTS

Variable	Values		
	Range	Mean	SD
Blood Sugar	60 - 321	152.7	58.7
Urea	16 – 70	35.9	10.5
CPK MB	26.7 – 49.7	36.7	4.8

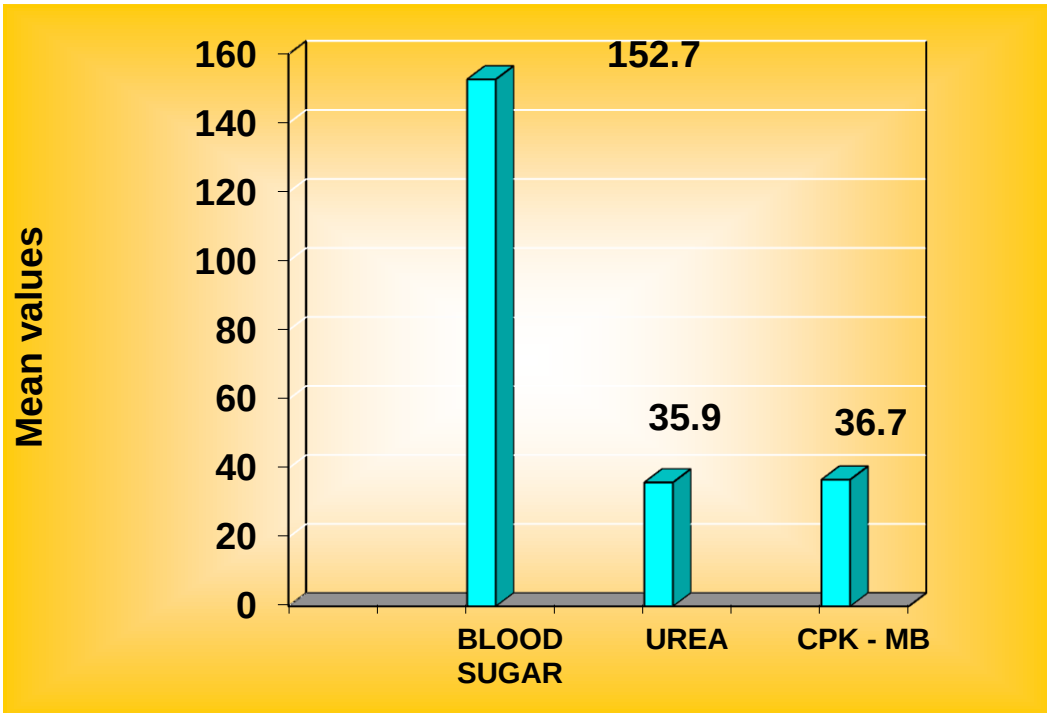

FIG.17 MEAN VALUES OF SUGAR/UREA/CPK-MB

LIPID PROFILE:

TABLE-14 LIPID PROFILE OF PATIENTS.

Variable	Normal Cases		Abnormal cases		Values		
	No.	%	No.	%	Range	Mean	SD
Total Cholesterol	188	94.0	12	6.0	85 – 246	164.1	28.3
HDL	13	6.5	187	93.5	16 – 48	32.3	6.5
LDL	183	91.5	17	8.5	50 – 168	104.5	22.0
TGL	199	99.5	1	0.5	60 - 204	137.3	33.6

The mean cholesterol values were 164 mg% with a range between 85 and 246mg%;mean HDL values being 32.3 mg%. The mean LDL value was around 104 mg% which is normal with a range between 50 and 168 mg%. The LDL cholesterol values were normal in majority of the individuals in this study with the HDL being low in majority of the individuals. The mean Triglyceride levels were around 137 mg%.

TOTAL CHOLESTEROL/ HDL /LDL / TGL

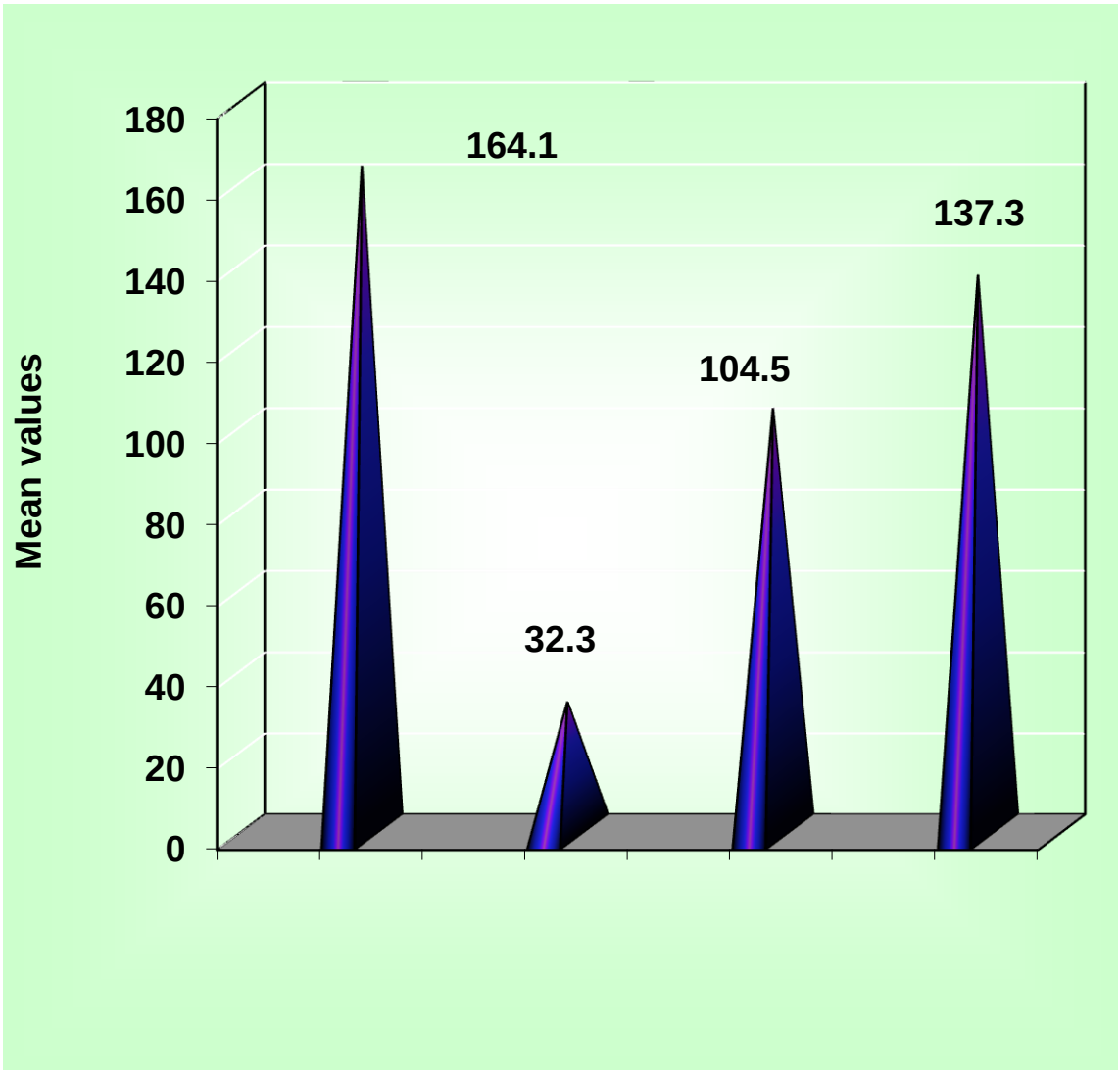

FIG.18 TOTAL CHOLESTEROL/ HDL /LDL / TGL

RISK FACTORS:

The risk factors were studied in all the 200 patients.

TABLE -15 RISK FACTORS AMONG PATIENTS

Risk Factors	Present		Absent	
	No.	%	No.	%
Smoking(among males- n=155)	120	77.4	35	22.6
Alcoholism(among males- n=155)	108	69.7	47	30.3
Physical Inactivity	96	48.0	104	52.0
Diabetes	74	37.0	126	63.0
Hypertension	66	33.0	134	67.0
Family History of CAD	65	32.5	135	67.5

Among the 155 males in the study, 77.4% were smokers and 69.7% consumed alcohol on a regular basis. None of the females had history of smoking and alcohol intake. Surprisingly only 37% in the group had diabetes. Hypertension was found in 33% of cases. Around 32.5% gave a positive family history of CAD.

Risk factors were different for Young MI (< 45 yrs). 91.7% were smokers, 88.9% consumed alcohol, around 50% had positive family history; 47% were leading a

sedentary life style and everyone 100% had low HDL. Around 16% were diabetic and 22% were hypertensive.

TABLE-16 RISK FACTORS IN YOUNG MI

RISK FACTORS	**Young age group (n=36)**	
	No	**%**
Smoking	33	91.7
Alcoholism	32	88.9
Physical inactivity	17	47.2
Diabetes	6	16.7
Hypertension	8	22.2
Family History	18	50.0
Abnormal HDL	36	100
Abnormal LDL	4	11.1
Diet Veg	1	2.8
Non Veg	36	97.2

TYPE OF MYOCARDIAL INFARCTION:

TABLE -17 MI TYPES AMONG PATIENTS:

Myocardial Infarction Type	**Cases**	
	No	**%**
Anterior wall	44	22
Antero lateral	3	1.5
Anteroseptal	29	14.5
Extensive Anterior Wall	29	14.5
Inferior wall	51	25.5
Inferoposterior wall	26	13.0
Lateral wall	8	4.0
Inferolateral&RVMI	10	5.0
Total	**200**	**200**

Among the MI types Inferior wall (25.5%) and Anterior wall (22%) were the most common types .Inferolateral, Anterolateral and Right Ventricular MI were comparatively less commonly observed in this study.

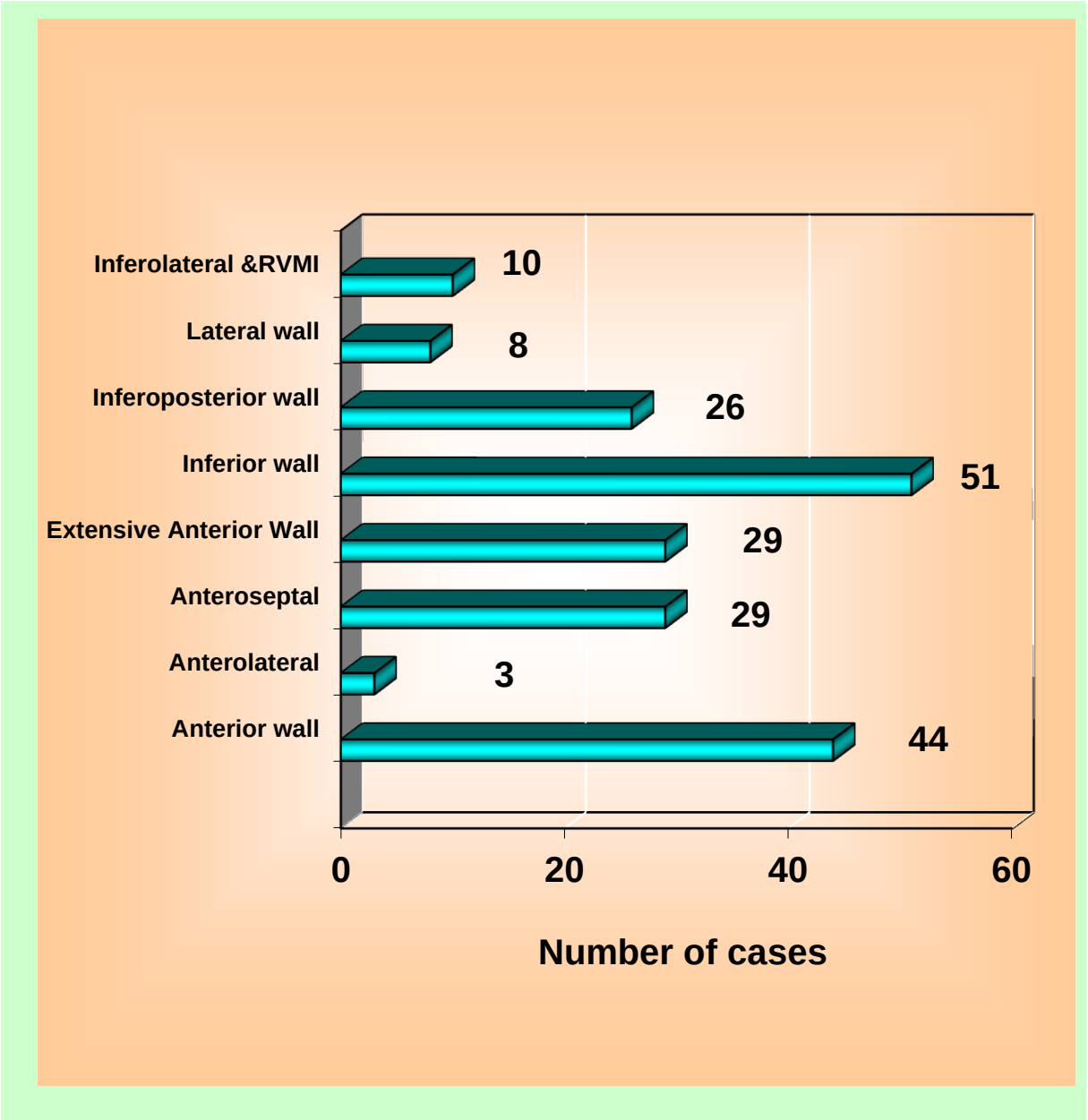

FIG.19 THE TYPES OF MI SEEN AMONG PATIENTS

TIMI RISK SCORE :

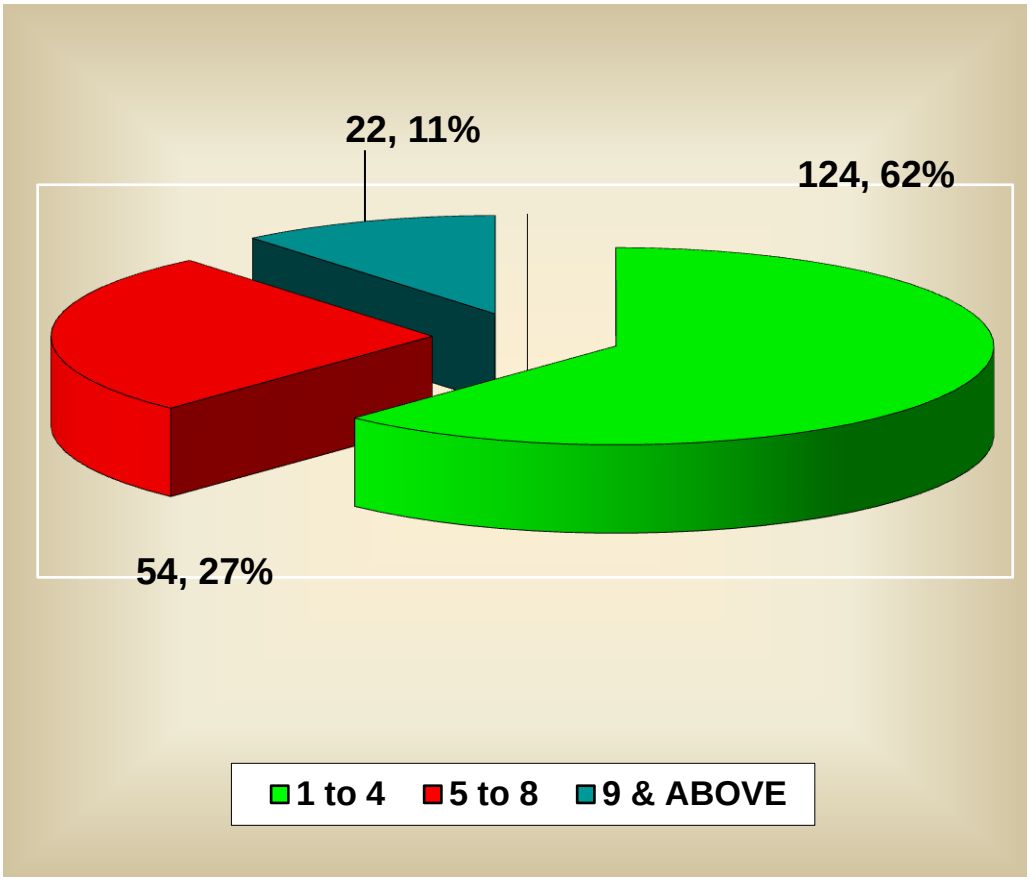

FIG.20 TIMI RISK SCORE

Among the 200 patients,124 cases (62%) had a TIMI Risk score of 1 to 4, 54 cases (27%) had a TIMI risk score of 5 to 8, 22 cases (11%) had a TIMI risk score of 9 and above.

TABLE-18 TIMI RISK SCORE

TIMI Score	Cases	
	No	%
1 - 4	124	62.0
5 - 8	54	27.0
9 & Above	22	11.0
Range	0 – 12	
Mean	4.29	
SD	2.7	

ECHO STUDY:

Among the 182 people in whom ECHO was done,36 cases (19.8%) had normal LV function,130 cases (71.4%) had mild LV dysfunction, 13 cases (7.1%) had moderate LV dysfunction and 3 cases (1.7%) .

TABLE -19 ECHO STUDY

Echo Result	Cases	
	No	%
Normal LV Function	36	19.8
Mild LV Dysfunction	130	71.4
Moderate Dysfunction	13	7.1
Severe Dysfunction	3	1.7
Total	182	100

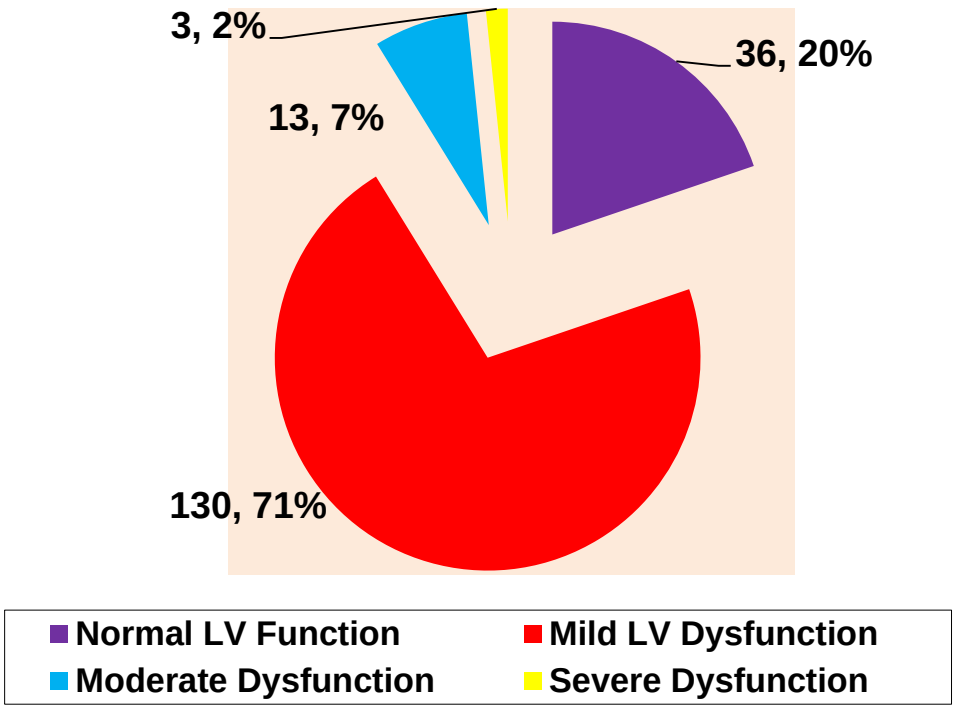

FIG. 21 ECHO RESULTS

OUTCOME:

TABLE-20 OUTCOME OF PATIENTS AT THE END OF FIRST WEEK

Out Come	Cases	
	No	%
Discharged alive	182	91.0
Death	18	9.0
Total	200	100

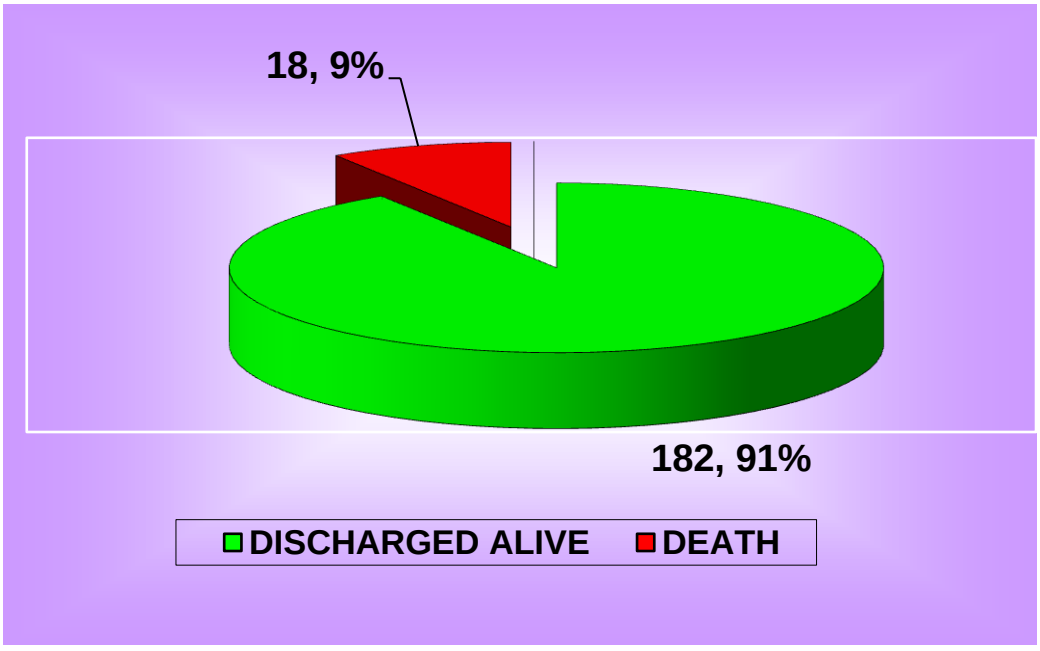

FIG.22 OUTCOME AT THE END OF ONE WEEK

TIMI RISK SCORE AND OUTCOME:

TABLE -21 RELATIONSHIP BETWEEN TIMI SCORE AND OUTCOME

TIMI risk score and Outcome **TIMI Risk Score**	**No of Cases**	**Out Come**			
		Discharged alive		**Death**	
		No	**%**	**No**	**%**
1 - 4	124	124	100	-	-
5 - 8	54	50	92.6	4	7.4
9 & Above	22	8	36.4	14	63.6
Mean Time Risk Score SD		3.8 2.25		9.28 1.67	
'p'		**<0.0001 Significant**			

All the patients (124 cases) who had a TIMI risk score of 1 to 4 survived at the end of first week, in the 54 patients with a TIMI risk score of 5 to 8, 50 patients survived at the end of first week. The percentage of mortality was 7.4%. In the 22 patients with a TIMI score of 9 and above, 14 expired at the end of first week with a mortality of 63.6%. The association is statistically significant.

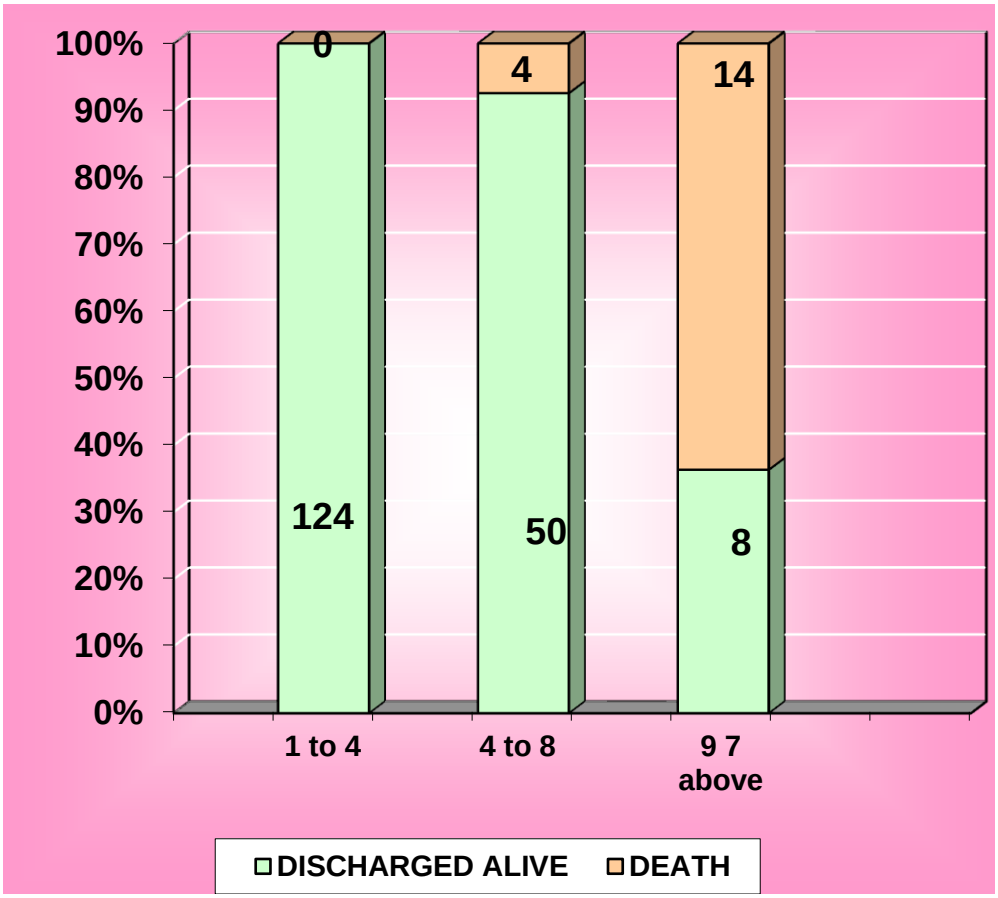

FIG.23 TIMI SCORE AND OUTCOME

DISCUSSION

DISCUSSION:

Coronary Artery Disease is an important cause of death in the world. The symptoms of Coronary artery disease vary from exertional chest pain due to Stable Angina to Sudden death due to ventricular fibrillation. Since data regarding risk factors has been largely been derived from developed countries this study has been done with regard to risk factors in the predominantly rural population.

Coronary artery Disease is more common among males. In our study, males accounted for 77.5% of the cases(155 cases) and females accounted for 22.5% of the cases(45 cases).The incidence of Coronary artery disease increases after the fourth decade of life. In our study, majority of the patients (68.5%) were above the age of fifty years. With regard to the presenting complaints, though chest pain was the most common presenting complaint in majority of individuals (67%), around 75% of the patients above 70 years of age, breathlessness and giddiness was the major presenting complaint. This may be due to the fact that elderly have poor pain perception and hence do not present with chest pain[67] .

Though the incidence of Myocardial Infarction is said to increase with each decade , in our study only 16.5 % of cases were above 70 years of age. Goyal et al[68] found that 52% of the Cardiovascular deaths in India were below 70 years. In our study 83.5% of cases were below 70 years. This may be due to the sample size

of the study. This might also be due to the fact that Elderly Patients usually have multiple co morbidities and may have expired before coming to a referral centre.

Patients with Coronary Artery disease have multiple risk factors both modifiable and non modifiable. Men above 45 years and Women above 55 years, Old age, positive family history, race are non-modifiable risk factors. Diabetes mellitus, hypertension, dyslipidemia, smoking, metabolic syndrome, physical inactivity appear to be modifiable risk factors.

Among the 155 males in the study, 77.4% were smokers and 69.7% gave history of excess alcohol intake. Excess consumption of tobacco in varied forms may be a leading cause of coronary artery disease in the lower socioeconomic group[69] . In our study, diabetes was found in 37% of the cases and hypertension in 33% of the cases. In our study, 41% belonged to the overweight category with just 6% belonging to the obesity category. This may be due to the fact that many patients belong to the poor socioeconomic status and also due to the fact that the patients may have had abdominal obesity. Positive family history was found in 32.5% of the cases.

In Young MI (<45 years), 91.7% were smokers, 88.9% consumed alcohol, around 50% had positive family history; 47% were leading a sedentary life style and everyone 100% had low HDL.

Inferior wall MI (25.5%) and Anterior wall MI (22%) were the most common types of MI that were seen. Among the 200 patients, 91% were discharged alive and 9% expired. The most common cause of death was cardiogenic shock. Among the 18 patients who expired, 14 patients had a Thrombolysis in Myocardial Infarction(TIMI) risk score of 9 and above and the remaining four had a TIMI score of 5 to 8. mortality was not seen in those with a TIMI risk score of below five. The association was statistically significant ($p<0.0001$).

Among the survivors around 72% of them had mild LV dysfunction, around 20% had normal LV function. 7.1% of patients had moderate LV dysfunction and 1.7% had severe LV dysfunction.

There may be many shortcomings in this study. But this study will definitely give us a fair idea of the risk factors for MI in the rural Indian population.

SUMMARY

SUMMARY:

In our study, it was found,

- Males were more commonly affected than females
- Age group above 50 years was most commonly affected
- Majority of individuals belonged to 51-60 years age bracket
- Chest pain was the most common presenting complaint, but in individuals above 70 years breathlessness and giddiness were more common
- Smoking and excess alcohol intake were the major contributors followed by diabetes and hypertension
- Inferior Wall MI and Anterior Wall MI were the most common MI types seen.
- Patients with higher TIMI risk score had increased mortality.

CONCLUSION

CONCLUSION:

Coronary Artery Disease continues to exact a heavy toll on the Indian population. Though significant strides have been made in the field of interventional cardiology like angioplasty, pacemakers, implantable cardioverter defibrillators, etc many of these advances are yet to reach the masses. The need of the hour is to implement strict preventive measures which include health education, legislative measures like banning smoking in public places and enforcing them strictly, emphasizing the importance of a healthy diet and an active lifestyle. These measures may help in the long run in reducing the burden of cardiovascular diseases in our country.

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BIBLIOGRAPHY:

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ANNEXURES

ANNEXURE-1

PROFORMA

CLINICAL PROFILE OF PATIENTS WITH MYOCARDIAL INFARCTION

PATIENT INFORMATION SHEET

NAME: AGE/SEX: IP NO:

DATE AND TIME OF ADMISSION:

PRESENTING COMPLAINTS:

PR: BP:

HT: WT: BMI:

ECG:

MI TYPE:

LIPID PROFILE: TC: HDL: LDL:

VLDL: TGL:

BLD SUGAR: UREA: CREATININE:

FAMILY HISTORY: CKD:

CARDIAC MARKERS: CPK-MB

ECHO:

CHAMBERS:

VALVES:

LV FUNCTION:

REGIONAL WALL MOTION ABNORMALITY:

IVS(d)/LVDd/LVDs/EF:

CLOT/PERICARDIAL EFFUSION:

RISK FACTORS:

SMOKING	
ALCOHOL	
FOOD HABITS	
OBESITY	
PHYSICAL INACTIVITY	
DIABETES	
HYPERTENSION	

TIMI RISK SCORE - STEMI

RISK FACTORS	POINTS
DM/HT/HISTORY OF ANGINA	
SYSTOLIC BP<100mm HG	
HEART RATE>100/MIN	
KILLIP CLASS II-IV	
BODY WT<67 KG	
ANTR ST ELEVATION/LBBB	
TIME TO TREAT > 4 HRS	
AGE > 75 YRS	
AGE 65-74 YRS	

ANNEXURE-2

MASTER CHART

CLINICAL PROFILE OF PATIENTS WITH MYOCARDIAL INFARCTION

S.NO	NAME	AGE /SEX	COMPLAINTS	PULSE	BP	MI TYPE	BMI	SMOKER	ALCOHOLIC	FOOD HABITS	OBESITY	PHYSICAL INACTIVITY	DIABETES	HYPERTENSION	FAMILY HISTORY OF CAD	SUGAR	UREA	CREATININE	TC/HDL/LDL/TGL	CPK MB	TIMI SCORE	ECHO	OUTCOME
1	MUTHU	37/M	CHEST PAIN	72	160/100	INFERIOR WALL	23.2	YES	YES	NON VEG	NO	NO	NO	YES	NO	119	34	0.7	131/34/66/151	29.6	3	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
2	KANAGARAJ	74/M	GIDDINESS	70	110/70	INFEROPOSTERIOR WALL	23	NO	NO	NON VEG	NO	YES	NO	NO	NO	98	24	0.8	120/24/76/106	30.5	4	MILD LV DYSFUNCTION/46%	DISCHARGED
3	VIJAYARAJ	47/M	CHEST PAIN,SWEATING	80	90/70	ANTERIOR WALL	21.7	YES	YES	NON VEG	NO	NO	NO	NO	YES	66	18	1	121/29/67/125	32.2	4	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
4	ESWARAN	60/M	BREATHLESSNESS	76	110/70	INFERIOR/RV MI	23.4	NO	NO	NON VEG	NO	NO	NO	NO	YES	109	34	1.5	166/30/120/83	33.3	2	MILD LV DYSFUNCTION/46%	DISCHARGED
5	MOOSA	57/M	GIDDINESS	94	120/80	INFERIOR WALL	24.4	NO	NO	NON VEG	NO	YES	NO	NO	YES	113	50	1.1	113/27/60/132	27.6	1	NORMAL LV FUNCTION/58%	DISCHARGED
6	MADASAMY	67/M	CHEST PAIN	76	140/100	EXTENSIVE ANTERIOR WALL	25.5	NO	NO	NON VEG	YES	YES	YES	YES	YES	160	22	0.9	130/16/98/80	28.4	8	MILD LV DYSFUNCTION/40%/LVH	DISCHARGED
7	SHEIKH	50/M	CHEST PAIN	98	160/100	EXTENSIVE ANTERIOR WALL	30.79	YES	YES	NON VEG	YES	YES	YES	YES	NO	116	40	0.8	180/40/108/160	29.9	3	MILD LV DYSFUNCTION/46%	DISCHARGED
8	SANKARNARAYAN	46/M	CHEST PAIN	106	160/110	INFERIOR WALL	22.83	YES	YES	NON VEG	NO	NO	YES	YES	NO	146	50	0.7	186/30/126/146	29.1	4	NORMAL LV FUNCTION/60%	DISCHARGED
9	SUNDARAMMAL	70/F	GIDDINESS	42	80/60	INFERIOR WALL	28.6	NO	NO	NON VEG	YES	YES	YES	YES	NO	188	41	1.1	188/46/110/160	38.6	9		DEATH
10	MURUGAN	45/M	CHEST PAIN	94	160/100	ANTERIOR WALL	23.24	YES	YES	NON VEG	NO	NO	NO	NO	NO	106	42	0.6	180/29/126/125	34.8	4	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
11	DEVAKI	60/F	CHEST PAIN	64	180/110	INFERIOR WALL	31.64	NO	NO	NON VEG	YES	YES	YES	YES	NO	146	34	0.6	200/40/130/146	32.7	3	NORMAL LV FUNCTION/54%/LVH	DISCHARGED
12	SAILAPPAN	77/M	BREATHLESSNESS	62	170/100	INFEROPOSTERIOR WALL	21.8	YES	YES	NON VEG	NO	NO	NO	YES	NO	133	37	1.1	188/42/126/100	37.5	6	NORMAL LV FUNCTION/54%/LVH	DISCHARGED
13	BALAKRISHNAN	28/M	CHEST PAIN	90	150/100	ANTERIOR WALL	29	YES	YES	NON VEG	YES	YES	NO	NO	NO	116	23	0.8	166/34/116/80	36.4	4	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
14	KANNAN	40/M	BREATHLESSNESS	96	150/100	ANTERIOR WALL	30.5	YES	YES	NON VEG	YES	YES	NO	NO	NO	96	19	0.9	160/32/102/136	32.2	3	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
15	RANGANATHAN	61/M	CHEST PAIN	106	160/100	EXTENSIVE ANTERIOR WALL	29.7	YES	YES	NON VEG	YES	YES	YES	NO	NO	186	47	1.1	196/40/116/200	40.9	7	MODERATE LV DYSFUNCTION/32%LVH	DISCHARGED
16	RAJASEKAR	52/M	CHEST PAIN	90	180/110	EXTENSIVE ANTERIOR WALL	30.4	YES	YES	NON VEG	YES	YES	NO	YES	YES	118	42	1	170/30/104/180	32.6	5	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
17	NARAYANAN	76/M	BREATHLESSNESS	104	180/110	ANTERIOR WALL	27.66	NO	NO	VEG	YES	NO	NO	YES	NO	130	58	1.3	184/32/112/194	33.7	10	MODERATE LV DYSFUNCTION/38%/LVH	DISCHARGED
18	POOMANI	58/F	CHEST PAIN	104	80/60	EXTENSIVE ANTERIOR WALL	31.6	NO	NO	NON VEG	YES	YES	YES	NO	NO	230	70	1.5	190/28/126/186	41.1	10		DEATH
19	LILY PUSHPAM	58/F	CHEST PAIN	62	160/100	INFEROPOSTERIOR WALL	31.6	NO	NO	VEG	YES	YES	NO	YES	NO	146	36	0.7	146/22/108/80	29.2	1	NORMAL LV FUNCTION/56%/LVH	DISCHARGED
20	NAMBIRAJAN	50/M	CHEST PAIN	94	180/100	EXTENSIVE ANTERIOR WALL	29.6	NO	NO	NON VEG	YES	YES	YES	NO	YES	198	40	1.3	174/30/114/156	39.4	5	MODERATE LV DYSFUNCTION/38%/LVH	DISCHARGED
21	PETCHIAMMAL	64/F	CHEST PAIN	106	160/90	EXTENSIVE ANTERIOR WALL	31.26	NO	NO	VEG	YES	YES	YES	NO	NO	174	41	1.2	136/30/78/144	30.3	9	MILD LV DYSFUNCTION/49%	DISCHARGED
22	PONNUSAMY	67/M	GIDDINESS	96	170/100	ANTEROSEPTAL	23.6	YES	NO	NON VEG	NO	NO	YES	NO	YES	194	60	1.4	144/22/84/190	32.1	7	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
23	SEETHALAKSHMI	72/F	CHEST PAIN	104	120/70	ANTERIOR WALL	35.15	NO	NO	NON VEG	YES	YES	YES	NO	NO	284	50	0.9	182/42/100/198	34.2	9	MODERATE LV DYSFUNCTION/32%LVH	DISCHARGED
24	MADAKANNU	71/M	BREATHLESSNESS	78	160/100	INFERIOR WALL	22.6	NO	NO	NON VEG	NO	NO	YES	NO	NO	180	36	1.4	194/36/124/174	29.8	3	NORMAL LV FUNCTION/52%/LVH	DISCHARGED
25	SUBRAMANIAN	62/M	GIDDINESS	64	120/70	INFERIOR WALL	28.2	NO	NO	NON VEG	YES	YES	YES	NO	NO	183	44	1.4	136/28/88/100	34.5	1	NORMAL LV FUNCTION/54%/LVH	DISCHARGED
26	GANESH	48/M	CHEST PAIN	74	130/70	INFERIOR WALL	21.22	YES	NO	NON VEG	NO	YES	NO	NO	NO	110	22	0.9	156/32/90/170	38.8	1	NORMAL LV FUNCTION/56%/LVH	DISCHARGED
27	MARIAPPAN	36/M	BREATHLESSNESS	103	160/110	ANTEROSEPTAL	22.15	YES	NO	NON VEG	NO	NO	NO	NO	NO	85	35	0.8	192/32/140/102	40.7	6	MILD LV DYSFUNCTION/48%	DISCHARGED
28	MOOKAMAL	75/F	CHEST PAIN	105	110/70	ANTERIOR WALL	29.1	NO	NO	VEG	YES	YES	YES	NO	NO	180	49	1.1	146/34/80/166	37.7	9	MILD LV DYSFUNCTION/45%	DISCHARGED
29	MURUGAIH	63/M	BREATHLESSNESS	80	110/70	ANTEROSEPTAL	22.7	YES	YES	NON VEG	NO	NO	NO	NO	YES	140	48	1.4	210/32/150/140	36.6	4	MILD LV DYSFUNCTION/48%	DISCHARGED
30	ANTONY JOSEPH	48/M	BREATHLESSNESS	98	160/90	EXTENSIVE ANTERIOR WALL	23.6	YES	YES	NON VEG	NO	NO	NO	YES	NO	106	46	1.4	184/40/120/120	43.8	6	SEVERE LV DYSFUNCTION/29%/LV DILATED	DISCHARGED
31	SELVARAJ	65/M	CHEST PAIN	48	100/60	INFEROPOSTERIOR WALL	23.1	YES	NO	NON VEG	NO	NO	NO	NO	YES	144	31	1.5	122/25/72/126	40.1	7	MILD LV DYSFUNCTION/43%	DISCHARGED
32	UCHIMAKALI	45/F	CHEST PAIN	90	150/90	INFERIOR WALL	23.9	NO	NO	NON VEG	NO	YES	YES	YES	NO	163	31	0.9	160/33/106/104	38.7	5	MILD LV DYSFUNCTION/45%	DISCHARGED
33	MURUGAN	53/M	BREATHLESSNESS	64	170/100	EXTENSIVE ANTERIOR WALL	23.3	YES	YES	NON VEG	NO	NO	NO	NO	YES	87	23	0.9	164/18/110/180	41.9	2	MODERATE LV DYSFUNCTION/32%LVH	DISCHARGED
34	RAJAMMAL	70/F	BREATHLESSNESS	96	160/90	ANTEROSEPTAL	22.8	NO	NO	NON VEG	NO	NO	NO	NO	NO	106	49	0.7	190/42/120/140	37.2	5	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
35	ARUMUGAM(D)	50/M	CHEST PAIN	108	90/60	ANTEROSEPTAL	22.2	YES	YES	NON VEG	NO	NO	NO	NO	NO	105	48	1.1	176/24/116/184	49.7	10		DEATH
36	VALAYAPANDIAN	55/M	GIDDINESS	78	150/100	INFEROPOSTERIOR WALL	21.2	NO	NO	NON VEG	NO	NO	NO	NO	NO	91	16	0.8	151/30/106/88	36.9	1	NORMAL LV FUNCTION/61%	DISCHARGED
37	CHELLADURAI	38/M	CHEST PAIN	86	160/100	ANTERIOR WALL	21.5	YES	YES	NON VEG	NO	YES	NO	NO	YES	109	30	1.4	172/38/116/90	34.9	2	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
38	SUBRAMANIAM	55/M	CHEST PAIN	94	180/100	ANTEROSEPTAL	22.1	YES	YES	VEG	NO	YES	NO	YES	YES	106	39	0.7	192/36/120/180	29.4	5	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
39	KANNAN	53/M	CHEST PAIN	75	150/90	ANTEROSEPTAL	22.6	YES	YES	NON VEG	NO	NO	NO	NO	YES	68	18	0.8	134/28/80/130	36.1	4	MILD LV DYSFUNCTION/40%	DISCHARGED
40	KALIMUTHU	47/M	GIDDINESS	56	100/70	INFERIOR WALL	25.9	YES	YES	NON VEG	YES	YES	YES	NO	NO	197	34	1.4	182/40/112/149	32.7	7	NORMAL LV FUNCTION/60%	DISCHARGED
41	RAHEEMA	63/F	CHEST PAIN	116	80/60	EXTENSIVE ANTERIOR WALL	29.9	NO	NO	NON VEG	YES	YES	YES	YES	NO	199	44	0.7	152/30/100/110	46.7	10		DEATH
42	CHANDRAMOHAN	37/M	GIDDINESS	80	140/90	INFERIOR WALL	26.2	YES	YES	NON VEG	YES	YES	NO	NO	YES	106	43	1.1	160/38/106/80	38.6	3	NORMAL LV FUNCTION/58%	DISCHARGED
43	NATHAN	54/M	CHEST PAIN	50	110/70	INFERIOR WALL	33.2	NO	NO	NON VEG	YES	YES	YES	NO	YES	307	24	1.4	210/29/160/107	30.7	4	MILD LV DYSFUNCTION/45%	DISCHARGED
44	MARIAMMAL	79/F	GIDDINESS	96	80/60	ANTERIOR WALL	28.6	NO	NO	NON VEG	YES	YES	YES	YES	NO	219	40	1.5	240/36/160/180	40.9	11		DEATH
45	KUMARAVEL	40/M	CHEST PAIN	82	140/90	ANTERIOR WALL	29.4	NO	NO	NON VEG	YES	YES	YES	YES	YES	248	23	1.2	199/39/140/104	36.7	4	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
46	NATCHATHIRAM	50/F	CHEST PAIN	90	130/90	EXTENSIVE ANTERIOR WALL	27.1	NO	NO	NON VEG	YES	NO	NO	NO	YES	101	28	0.6	103/22/61/99	38.6	4	MODERATE LV DYSFUNCTION/32%LVH	DISCHARGED
47	MARIAPPAN	55/M	CHEST PAIN	80	100/80	INFEROPOSTERIOR WALL	32	YES	YES	NON VEG	YES	YES	YES	NO	YES	217	17	1.2	126/22/70/174	39.7	4	MILD LV DYSFUNCTION/47%/LVH	DISCHARGED
48	PERUMAL	57/M	CHEST PAIN	62	110/60	INFERIOR WALL	27.2	YES	YES	NON VEG	YES	YES	NO	YES	NO	270	23	0.9	128/30/70/142	39.1	2	NORMAL LV FUNCTION/56%	DISCHARGED
49	ANANTHRAM(D)	55/M	CHEST PAIN	108	90/60	ANTERIOR WALL	22.8	YES	YES	VEG	NO	NO	NO	NO	YES	98	52	1.3	180/21/123/190	36.5	11		DEATH
50	MARIAKUTTY	39/M	CHEST PAIN	66	140/90	INFERIOR WALL	28.3	YES	YES	NON VEG	YES	YES	NO	NO	NO	147	25	1.2	108/24/68/145	29.9	1	NORMAL LV FUNCTION/61%	DISCHARGED
51	AYYADURAI	34/M	BREATHLESSNESS	70	100/70	EXTENSIVE ANTERIOR WALL	27.7	YES	YES	NON VEG	YES	YES	NO	NO	YES	80	21	1	116/20/80/84	30.2	5	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
52	NATARAJAN	65/M	BREATHLESSNESS	78	120/80	ANTEROSEPTAL	28.7	NO	NO	NON VEG	YES	YES	NO	NO	NO	97	20	0.7	85/16/54/76	34.3	3	MILD LV DYSFUNCTION/45%	DISCHARGED
53	JAGADHEESH	40/M	CHEST PAIN	75	130/90	INFEROPOSTERIOR WALL	27.6	YES	YES	NON VEG	YES	NO	NO	NO	YES	210	16	0.9	186/34/131/113	33.8	0	NORMAL LV FUNCTION/56%	DISCHARGED
54	JEYARAJ(D)	56/M	CHEST PAIN	60	80/60	EXTENSIVE ANTERIOR WALL	26.9	YES	YES	NON VEG	YES	YES	YES	NO	YES	260	26	1.4	133/24/70/196	42.4	7		DEATH
55	CHELLIAH	64/M	CHEST PAIN	78	130/70	INFEROPOSTERIOR WALL	27.3	YES	YES	NON VEG	YES	YES	NO	NO	NO	159	34	1.5	134/32/80/110	37.9	1	NORMAL LV FUNCTION/56%/LVH	DISCHARGED
56	BEER MYDEEEN	34/M	CHEST PAIN	66	110/70	INFERIOR WALL	27.6	YES	YES	NON VEG	YES	YES	NO	NO	NO	136	16	0.8	172/38/108/80	33.3	1	MILD LV DYSFUNCTION/49%	DISCHARGED
57	PERUMAL	65/F	CHEST PAIN	96	180/100	ANTERIOR WALL	29.4	NO	NO	NON VEG	YES	YES	NO	NO	NO	69	24	0.9	166/30/116/98	38.4	6	MILD LV DYSFUNCTION/45%	DISCHARGED
58	RAJESHWARI	52/F	CHEST PAIN	92	170/100	LATERAL WALL	28.8	NO	NO	NON VEG	YES	YES	NO	YES	NO	124	18	0.6	166/26/100/196	28.7	4	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
59	SINGARAJ	45/M	BREATHLESSNESS	86	120/80	ANTEROSEPTAL	27.1	YES	YES	NON VEG	YES	YES	YES	NO	YES	171	19	1	100/29/50/102	29.1	4	MILD LV DYSFUNCTION/45%	DISCHARGED
60	MUTHAIAH	75/M	BREATHLESSNESS	88	110/60	ANTERIOR WALL	27.8	YES	YES	NON VEG	YES	NO	NO	NO	NO	88	37	1.3	190/38/126/130	35.7	7	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
61	PETCHI	60/M	GIDDINESS	78	130/80	INFERIOR/RVMI	23.3	YES	YES	NON VEG	NO	NO	YES	YES	NO	160	29	1	138/26/78/173	35.5	1	NORMAL LV FUNCTION/56%	DISCHARGED
62	KOMBAN	40/M	CHEST PAIN	82	190/130	EXTENSIVE ANTERIOR WALL	24.9	NO	NO	NON VEG	NO	NO	NO	YES	YES	138	49	1.5	180/38/110/160	43.9	5	MODERATE LV DYSFUNCTION/32%LVH	DISCHARGED
63	ESAKKI	49/M	CHEST PAIN	80	120/90	INFERIOR WALL	24.6	YES	YES	NON VEG	NO	NO	NO	NO	YES	60	24	1.1	134/24/92/90	36.8	1	MILD LV DYSFUNCTION/49%	DISCHARGED
64	MEENAMMAL(D)	55/F	CHEST PAIN	104	80/50	EXTENSIVE ANTERIOR WALL	28.8	NO	NO	NON VEG	YES	YES	YES	NO	NO	186	50	1.3	216/36/140/200	48.8	10		DEATH
65	PACKIARAJ	76/M	GIDDINESS	70	130/80	INFERIOR WALL	24.4	YES	NO	NON VEG	NO	NO	NO	NO	NO	144	40	1	148/30/90/140	34.5	4	MILD LV DYSFUNCTION/44%	DISCHARGED
66	MARIAPPAN	59/M	CHEST PAIN	106	130/90	LATERAL WALL	24.5	YES	YES	NON VEG	NO	NO	NO	NO	NO	116	30	0.8	106/20/74/60	32.7	7	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
67	SUDALIMUTHU	47/M	CHEST PAIN	88	110/70	EXTENSIVE ANTERIOR WALL	24.2	YES	NO	NON VEG	NO	NO	NO		YES	110	43	1.2	108/28/60/100	39.7	3	MILD LV DYSFUNCTION/49%	DISCHARGED
68	RAMAN	75/M	GIDDINESS	78	140/100	HIGH LATERAL MI	23.8	YES	YES	NON VEG	NO	NO	NO	NO	NO	157	42	1.5	139/39/76/136	35.6	3	MILD LV DYSFUNCTION/45%	DISCHARGED
69	PALAVESAM	54/M	BREATHLESSNESS	86	130/70	ANTEROSEPTAL	24.9	NO	NO	VEG	NO	NO	NO	NO	YES	102	26	1.1	120/36/64/100	35.9	1	NORMAL LV FUNCTION/56%	DISCHARGED
70	GANESAN	40/M	BREATHLESSNESS	60	130/90	ANTEROLATERAL	24.7	NO	YES	VEG	NO	NO	NO	NO	NO	114	18	0.7	156/36/95/124	39.8	2	MILD LV DYSFUNCTION/49%	DISCHARGED
71	SELVARAJ(D)	74/M	GIDDINESS	54	80/60	INFEROPOSTERIOR WALL	23.2	YES	YES	NON VEG	NO	YES	YES	NO	NO	236	52	1.1	126/26/80/104	47.5	9		DEATH
72	MASOODH	63/M	CHEST PAIN	64	160/100	INFERIOR WALL	24.7	YES	NO	NON VEG	NO	NO	NO	NO	YES	188	49	1	123/20/83/106	30.6	1	NORMAL LV FUNCTION/69%	DISCHARGED
73	BEER MOHAMMED	64/M	CHEST PAIN	60	120/70	INFERIOR WALL	24.6	YES	NO	NON VEG	NO	YES	YES	YES	YES	160	39	1.3	156/25/110/104	35.5	2	NORMAL LV FUNCTION/57%	DISCHARGED
74	MARIYA ANTONY	56/M	GIDDINESS	66	160/100	INFERIOR WALL	24.4	NO	YES	NON VEG	NO	NO	YES	NO	NO	242	33	1.1	160/26/102/168	26.7	1	NORMAL LV FUNCTION/61%	DISCHARGED
75	MOHD.ABU BAKR	39/M	CHEST PAIN	96	150/100	ANTEROSEPTAL	24.7	YES	YES	NON VEG	NO	YES	NO	NO	YES	147	41	1.3	218/38/144/180	33.6	1	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
76	THALAVAI	75/M	BREATHLESSNESS	94	170/110	ANTERIOR WALL	24.4	NO	NO	NON VEG	NO	NO	NO	YES	NO	94	44	0.7	146/30/100/80	34.7	5	NORMAL LV FUNCTION/62%/LVH	DISCHARGED
77	RAMASAMY	68/M	CHEST PAIN	68	100/70	INFEROPOSTERIOR WALL	24.7	YES	YES	NON VEG	NO	NO	NO	YES	YES	96	38	1.1	168/33/110/126	39.1	5	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
78	VELAMMAL	48/F	CHEST PAIN	54	90/60	INFEROPOSTERIOR WALL	31.1	NO	NO	VEG	YES	YES	YES	NO	NO	149	50	1.1	166/36/90/200	43.2	7		DEATH
79	PALANI SELVAM	55/M	CHEST PAIN	78	160/100	EXTENSIVE ANTERIOR WALL	24.1	YES	NO	NON VEG	NO	NO	NO	NO	YES	126	18	1.3	123/25/66/161	39.7	1	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
80	PALAVESAM	61/M	GIDDINESS	66	140/70	INFERIOR WALL	24.8	NO	NO	VEG	NO	NO	NO	NO	YES	101	25	1.2	133/31/80/111	34.6	1	NORMAL LV FUNCTION/62%/LVH	DISCHARGED
81	THANGAVEL	46/M	CHEST PAIN	90	150/100	ANTERIOR WALL	24.8	YES	YES	NON VEG	NO	YES	NO	NO	NO	73	20	0.9	136/32/80/120	40.5	4	MILD LV DYSFUNCTION/49%	DISCHARGED
82	VELSAMY	55/M	CHEST PAIN	86	140/80	INFEROPOSTERIOR WALL	24.9	YES	YES	NON VEG	NO	NO	NO	NO	YES	94	24	0.6	97/20/59/89	30.9	1	NORMAL LV FUNCTION/58%	DISCHARGED
83	PURUSHOTHAMAN	70/M	BREATHLESSNESS	83	100/60	INFERIOR WALL	24.1	YES	YES	NON VEG	NO	NO	NO	NO	NO	216	40	1.2	225/41/144/198	37.6	7	MILD LV DYSFUNCTION/45%	DISCHARGED
84	KOVILPICHAI	50/M	BREATHLESSNESS	98	160/100	ANTEROLATERAL	23.1	YES	YES	NON VEG	NO	YES	NO	NO	NO	90	33	1.2	136/26/84/130	36.8	3	MILD LV DYSFUNCTION/46%	DISCHARGED
85	SUBRAMANIYAN	84/M	CHEST PAIN	60	170/110	INFERIOR WALL	21.9	YES	YES	NON VEG	NO	NO	YES	YES	NO	186	53	1.5	130/30/70/154	43.6	5	MODERATE LV DYSFUNCTION/32%LVH	DISCHARGED
86	MILTON	38/M	CHEST PAIN	110	90/60	EXTENSIVE ANTERIOR WALL	27.1	YES	YES	NON VEG	YES	YES	NO	NO	NO	144	58	1.2	164/39/100/130	46.8	9		DEATH
87	BAGAVATHY	55/F	CHEST PAIN	94	180/110	ANTERIOR WALL	27.9	NO	NO	NON VEG	YES	YES	YES	YES	NO	266	54	1.1	186/36/120/153	35.5	3	MILD LV DYSFUNCTION/49%	DISCHARGED
88	KANNAN	63/M	GIDDINESS	74	150/110	INFEROPOSTERIOR WALL	22.8	YES	YES	NON VEG	NO	YES	NO	YES	NO	126	32	1.3	152/22/110/104	34.9	2	NORMAL LV FUNCTION/58%/LVH	DISCHARGED
89	MARIMUTHU	70/M	GIDDINESS	56	190/100	INFERIOR WALL	21.8	YES	NO	NON VEG	NO	NO	YES	YES	NO	170	56	0.9	135/30/76/96	39.5	4	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
90	SORNAM	68/F	CHEST PAIN	94	180/100	ANTERIOR WALL	28.8	NO	NO	NON VEG	YES	YES	YES	YES	YES	310	46	1.3	160/34/110/86	40.5	4	MILD LV DYSFUNCTION/49%	DISCHARGED
91	PERUMAL	45/M	GIDDINESS	64	140/100	INFEROPOSTERIOR WALL	21.4	YES	YES	NON VEG	NO	YES	NO	NO	NO	110	41	0.9	140/26/88/130	32.3	1	NORMAL LV FUNCTION/60%	DISCHARGED
92	MURUGESAN	46/M	CHEST PAIN	106	160/100	ANTERIOR WALL	29.3	YES	YES	NON VEG	YES	YES	NO	NO	NO	160	36	0.7	180/38/112/146	37.6	6	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
93	SIVABALAN	70/M	GIDDINESS	70	170/100	INFERIOR WALL	21.5	YES	YES	NON VEG	NO	NO	NO	YES	NO	106	30	0.9	140/30/88/110	38.7	3	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
94	SAMSUDEEN	37/M	BREATHLESSNESS	104	150/100	EXTENSIVE ANTERIOR WALL	26.9	YES	YES	NON VEG	YES	NO	NO	NO	YES	116	47	1.2	170/28/120/110	40.6	3	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
95	NALLAKANNU	35/M	CHEST PAIN	84	140/90	ANTERIOR WALL	26.6	YES	YES	NON VEG	YES	YES	NO	NO	NO	132	32	1.3	130/30/64/180	30.2	2	MILD LV DYSFUNCTION/49%	DISCHARGED
96	CHINNATHAMBI	47/M	CHEST PAIN	78	160/100	ANTEROSEPTAL	26.1	YES	YES	NON VEG	YES	YES	NO	YES	YES	170	36	1.3	182/36/116/150	38.6	3	MILD LV DYSFUNCTION/46%	DISCHARGED
97	ESAKKIPANDI	59/M	CHEST PAIN	84	110/70	INFERIOR WALL	23.1	YES	YES	NON VEG	NO	NO	YES	NO	YES	212	36	1.3	130/30/74/130	29.5	3	MILD LV DYSFUNCTION/49%	DISCHARGED
98	PALAVESAMUTHU	75/M	GIDDINESS	86	110/80	INFERIOR WALL	27.3	YES	YES	NON VEG	YES	YES	NO	NO	NO	156	34	1.2	162/38/90/170	30.1	4	MILD LV DYSFUNCTION/44%	DISCHARGED
99	BALASUBBU	53/M	CHEST PAIN	68	110/80	INFEROPOSTERIOR WALL	28.6	YES	YES	NON VEG	YES	YES	YES	NO	NO	270	36	1.4	178/38/104/180	39.9	3	MILD LV DYSFUNCTION/45%	DISCHARGED
100	ESAKKI	60/M	CHEST PAIN	78	140/90	ANTERIOR WALL	26.5	YES	YES	NON VEG	YES	YES	NO	NO	NO	89	35	1.1	146/36/92/90	40.5	3	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
101	SHANMUGAMAL	65/F	CHEST PAIN	76	160/110	ANTERIOR WALL	28.9	NO	NO	NON VEG	YES	YES	YES	NO	YES	284	40	1.3	172/30/108/170	39.4	5	MILD LV DYSFUNCTION/46%	DISCHARGED
102	SANKAR	70/M	GIDDINESS	68	140/90	ANTEROSEPTAL	22.1	YES	YES	NON VEG	NO	NO	NO	NO	NO	116	42	0.9	164/30/108/132	29.9	7	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
103	ESAKKIAMMAL	75/F	GIDDINESS	64	170/110	INFERIOR RVMI	26.7	NO	NO	NON VEG	YES	YES	NO	YES	NO	146	44	1.4	182/34/118/148	40.7	7	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
104	JANAKI	55/F	CHEST PAIN	86	170/100	EXTENSIVE ANTERIOR WALL	21.9	NO	NO	VEG	NO	NO	YES	YES	NO	250	41	1.4	150/30/92/140	33.8	4	MILD LV DYSFUNCTION/45%	DISCHARGED
105	MUTHUSAMY	55/M	CHEST PAIN	68	110/60	INFERIOR WALL	28.1	NO	YES	NON VEG	YES	YES	NO	NO	YES	152	42	1.3	200/40/136/120	38.9	1	MILD LV DYSFUNCTION/46%	DISCHARGED
106	MUTHU	56/M	GIDDINESS	74	120/70	INFERIOR/RVMI	28.6	YES	YES	NON VEG	YES	YES	YES	YES	YES	252	40	1.2	164/30/108/134	39.7	2	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
107	PETCHIKUTTI	35/M	CHEST PAIN	84	140/80	ANTERIOR WALL	26.3	YES	YES	NON VEG	YES	NO	YES	YES	YES	272	46	1.3	180/38/108/170	34.5	4	MILD LV DYSFUNCTION/48%	DISCHARGED
108	NAINAR	50/M	CHEST PAIN	106	80/60	EXTENSIVE ANTERIOR WALL	29.1	YES	YES	NON VEG	YES	YES	YES	NO	NO	286	70	1.1	184/26/118/204	46.6	10		DEATH
109	RAMASWAMY	76/M	CHEST PAIN	84	110/80	INFEROPOSTERIOR WALL	28.5	YES	YES	NON VEG	YES	NO	NO	NO	YES	202	40	1.2	178/40/102/180	40.4	5	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
110	MANONMANI	60/F	GIDDINESS	90	120/80	INFEROPOSTERIOR WALL	21.8	NO	NO	NON VEG	NO	NO	NO	NO	YES	146	40	1.1	146/30/80/180	30.5	2	MILD LV DYSFUNCTION/46%	DISCHARGED
111	ISHA	45/M	CHEST PAIN	82	150/100	ANTERIOR WALL	21.6	YES	YES	NON VEG	NO	NO	NO	YES	YES	165	32	0.8	126/27/83/80	39.5	3	MILD LV DYSFUNCTION/46%/LVH	DISCHARGED
112	ABDUL KADHAR	55/M	CHEST PAIN	76	120/70	ANTEROSEPTAL	21.7	YES	YES	NON VEG	NO	NO	YES	NO	NO	194	30	1.2	166/26/120/104	38.6	4	MILD LV DYSFUNCTION/49%/LVH	DISCHARGED
113	THIRUMALAI	50/F	CHEST PAIN	103	160/100	ANTERIOR WALL	29.3	YES	YES	NON VEG	YES	YES	YES	YES	NO	132	50	0.7	160/27/103/146	49.5	5		DEATH
114	SAHUL HAMEED	75/M	GIDDINESS	60	90/60	INFERIOR	23.9	YES	YES	NON VEG	NO	NO	NO	NO	NO	116	39	0.8	174/30/128/84	34.4	9	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
115	PARAMASIVAN	47/M	CHEST PAIN	110	100/60	EXTENSIVE ANTERIOR WALL	22.3	YES	YES	NON VEG	NO	NO	NO	NO	NO	111	46	0.8	184/30/126/136	43.3	9	SEVERE LV DYSFUNCTION/25%/LV DILATED	DISCHARGED
116	GURUSAMY	45/M	CHEST PAIN	106	86/60	ANTEROLATERAL	26.2	YES	YES	NON VEG	YES	YES	NO	NO	YES	142	30	0.9	154/31/100/116	46.1	10		DEATH
117	CHELLAIAH	60/M	GIDDINESS	72	130/80	INFEROPOSTERIOR WALL	21.8	YES	YES	NON VEG	NO	NO	NO	NO	NO	130	24	0.8	190/32/132/136	28.4	2	NORMAL LV FUNCTION/58%	DISCHARGED
118	ELISHA JACOB	53/F	CHEST PAIN	108	96/60	ANTERIOR WALL	27.7	NO	NO	VEG	YES	YES	YES	NO	NO	266	42	1.4	182/38/120/122	40.4	8		DEATH
119	PALPANDI	75/M	CHEST PAIN	62	180/100	INFEROPOSTERIOR WALL	22.2	YES	YES	NON VEG	NO	NO	NO	YES	NO	166	31	0.9	146/32/94/104	30.4	5	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
120	PADMAVATHY	56/F	CHEST PAIN	96	160/100	ANTERIOR WALL	28.6	NO	NO	VEG	YES	YES	YES	YES	NO	290	56	0.9	208/40/128/199	30.6	5	MILD LV DYSFUNCTION/46%/LVH	DISCHARGED
121	RATHINAM	70/M	GIDDINESS	66	100/60	INFERIOR WALL	21.7	YES	NO	NON VEG	NO	YES	NO	NO	NO	122	38	1.2	146/24/98/118	48.8	9		DEATH
122	GANAPATHY	70/M	CHEST PAIN	98	180/100	EXTENSIVE ANTERIOR WALL	21.9	YES	YES	NON VEG	NO	NO	NO	YES	NO	146	36	0.9	184/36/126/110	45.4	6	SEVERE LV DYSFUNCTION/24%/LVDILATED	DISCHARGED
123	KRISHNASAMY	51/M	GIDDINESS	90	160/100	INFEROPOSTERIOR WALL	28.4	NO	NO	VEG	YES	YES	NO	YES	NO	121	19	0.7	160/24/110/130	33.1	1	MILD LV DYSFUNCTION/49%	DISCHARGED
124	MOOKAN	70/M	CHEST PAIN	110	90/60	ANTERIOR WALL	29.8	YES	YES	NON VEG	YES	NO	YES	NO	NO	166	38	1.4	166/36/106/120	49.4	12		DEATH
125	CHITHRAKANI	58/M	CHEST PAIN	102	90/60	EXTENSIVE ANTERIOR WALL	26.9	YES	YES	NON VEG	YES	YES	NO	NO	YES	190	40	0.7	164/30/108/130	45.7	10		DEATH
126	NAGOORMESHA	60/M	BREATHLESSNESS	62	130/70	INFEROPOSTERIOR WALL	27.3	NO	NO	NON VEG	YES	YES	NO	NO	YES	261	41	1.3	184/38/116/136	34.4	2	MILD LV DYSFUNCTION/44%	DISCHARGED
127	PALKANI	64/M	GIDDINESS	90	150/90	INFERIOR/RV MI	28.2	NO	NO	NON VEG	YES	YES	NO	NO	NO	90	45	0.9	174/38/108/138	36.2	1	NORMAL LV FUNCTION/56%	DISCHARGED
128	UDAIYAR	75/M	BREATHLESSNESS	90	180/100	ANTERIOR WALL	22.6	NO	NO	VEG	NO	NO	NO	YES	NO	74	23	0.9	140/20/96/124	30.7	8	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
129	GANAPATHY	72/M	CHEST PAIN	94	140/80	ANTEROSEPTAL	21.8	NO	NO	VEG	NO	NO	NO	NO	NO	110	20	0.9	148/34/92/110	37.6	4	MILD LV DYSFUNCTION/48%	DISCHARGED
130	ESAKKI	42/M	CHEST PAIN	92	150/90	ANTEROSEPTAL	23.3	YES	YES	NON VEG	NO	NO	NO	NO	NO	111	40	0.9	146/28/92/130	39.8	3	MILD LV DYSFUNCTION/48%	DISCHARGED
131	GNAKKAN	73/M	CHEST PAIN	90	100/90	INFERIOR WALL	22.9	YES	YES	NON VEG	NO	NO	NO	YES	NO	160	19	1.1	184/36/106/136	39.1	5	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
132	VELAPPAN	72/M	GIDDINESS	98	160/100	EXTENSIVE ANTERIOR WALL	24.2	YES	YES	NON VEG	NO	NO	NO	YES	NO	116	33	0.9	196/38/140/138	39.5	6	MILD LV DYSFUNCTION/44%	DISCHARGED
133	PERUMAL	45/M	CHEST PAIN	84	150/90	ANTERIOR WALL	29.4	YES	YES	NON VEG	YES	NO	YES	NO	NO	272	38	1.5	190/38/118/170	38.7	3	MILD LV DYSFUNCTION/48%	DISCHARGED
134	INNARIMUTHU	64/M	CHEST PAIN	84	110/70	INFERIOR WALL	27.7	YES	YES	NON VEG	YES	YES	NO	NO	YES	121	36	1.2	190/40/114/180	35.6	1	MILD LV DYSFUNCTION/48%	DISCHARGED
135	PANDI	45/M	CHEST PAIN	90	160/90	ANTERIOR WALL	20.6	YES	YES	NON VEG	NO	NO	YES	YES	YES	321	42	1.2	180/28/120/160	39.6	4	MILD LV DYSFUNCTION/50%	DISCHARGED
136	MUPPIDATHY	65/M	CHEST PAIN	94	180/110	INFERIOR WALL	23.6	YES	YES	NON VEG	NO	NO	NO	NO	NO	116	46	1	178/38/110/146	30.6	4	MILD LV DYSFUNCTION/48%	DISCHARGED
137	ABDUL KADAR	53/M	CHEST PAIN	76	140/90	ANTEROSEPTAL	28.4	YES	YES	NON VEG	YES	YES	NO	NO	NO	146	34	0.9	164/42/87/188	40.7	3	MILD LV DYSFUNCTION/48%	DISCHARGED
138	PETCHIAPPAN	45/M	CHEST PAIN	96	170/90	ANTEROSEPTAL	23.6	YES	YES	NON VEG	NO	NO	NO	NO	NO	116	42	1.1	174/42/104/140	33.5	4	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
139	KOILAMMAL	55/F	CHEST PAIN	90	160/90	EXTENSIVE ANTERIOR WALL	27.4	NO	NO	NON VEG	YES	YES	YES	NO	NO	164	36	1.2	190/38/120/160	34.6	4	MILD LV DYSFUNCTION/40%/LVH	DISCHARGED
140	SUMITHA	50/F	CHEST PAIN	64	110/60	INFERIOR WALL	28.4	NO	NO	NON VEG	YES	YES	YES	NO	NO	269	47	1.1	180/46/110/120	29.6	2	MILD LV DYSFUNCTION/48%	DISCHARGED
141	ARUMAIRAJ	69/M	CHEST PAIN	106	160/100	ANTERIOR WALL	28.6	YES	NO	NON VEG	YES	YES	NO	YES	NO	116	42	1.6	188/38/118/160	40.2	8	MODERATE LV DYSFUNCTION/32%/LVH	DISCHARGED
142	RAJU	75/M	GIDDINESS	94	180/100	ANTERIOR WALL	24.8	NO	YES	NON VEG	NO	NO	YES	YES	NO	188	46	0.9	144/36/80/140	34.5	7	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
143	SARASWATHI	72/F	BREATHLESSNESS	68	180/100	INFERIOR WALL	29.4	NO	NO	VEG	YES	YES	YES	YES	NO	246	46	1.2	160/28/102/150	35.7	6	MILD LV DYSFUNCTION/46%/LVH	DISCHARGED
144	AYANUSAMY	54/M	CHEST PAIN	96	150/90	ANTEROSEPTAL	23.6	YES	YES	NON VEG	NO	NO	NO	NO	NO	106	29	0.7	130/28/82/100	35.9	1	MILD LV DYSFUNCTION/46%	DISCHARGED
145	SUDALAI	40/M	CHEST PAIN	104	110/50	EXTENSIVE ANTERIOR WALL	23.9	YES	NO	NON VEG	NO	NO	NO	NO	NO	76	31	1.1	198/38/130/150	38.7	6	MILD LV DYSFUNCTION / 40%/LV DILATED	DISCHARGED
146	SEEMAN	50/M	CHEST PAIN	84	140/90	LATERAL WALL	24.6	YES	YES	NON VEG	NO	NO	NO	NO	NO	102	16	0.7	164/32/110/110	36.8	1	NORMAL/54%	DISCHARGED
147	VEERABAGU	70/M	CHEST PAIN	104	100/60	ANTERIOR WALL	22.8	NO	NO	VEG	NO	NO	YES	NO	NO	174	30	0.9	130/18/92/100	41.1	12	MODERATE LV DYSFUNCTION/36%/LVH	DISCHARGED
148	BASKARAN	68/M	CHEST PAIN	90	160/100	INFERIOR WALL	29.6	YES	NO	NON VEG	YES	YES	NO	NO	NO	102	37	0.9	186/34/123/146	34.5	4	MILD LV DYSFUNCTION/47%/LVH	DISCHARGED
149	SAROJA	67/F	GIDDINESS	68	140/90	INFERIOR WALL	27.3	NO	NO	VEG	YES	YES	YES	NO	NO	269	41	1.1	240/46/154/190	37.4	4	MILD LV DYSFUNCTION/42%	DISCHARGED
150	KAJAMYDEEN	47/M	CHEST PAIN	60	130/80	INFERIOR WALL	22.1	YES	NO	NON VEG	NO	NO	YES	NO	YES	184	48	1.2	180/40/112/140	37.9	1	NORMAL/58.1%	DISCHARGED
151	PAVANASAM	55/M	CHEST PAIN	64	130/80	POSTERIOR WALL	22.7	YES	YES	NON VEG	NO	NO	NO	NO	NO	122	21	0.6	148/30/98/108	36.8	1	NORMAL/56%	DISCHARGED
152	MUPPIDATHY	55/F	CHEST PAIN	64	150/80	ANTEROSEPTAL	22.1	NO	NO	VEG	NO	NO	YES	YES	YES	136	41	1.1	138/38/80/106	29.6	4	MILD LV DYSFUNCTION/46%/LVH	DISCHARGED
153	ANNAMAL	60/F	CHEST PAIN	64	140/80	INFEROPOSTERIOR WALL	27.6	NO	NO	NON VEG	YES	YES	NO	NO	NO	116	31	0.8	144/28/96/102	36.9	3	MILD LV DYSFUNCTION/48%	DISCHARGED
154	RAMASAMY	45/M	CHEST PAIN	92	160/90	LATERAL WALL	22.8	YES	YES	NON VEG	NO	NO	NO	YES	YES	141	34	0.7	134/23/90/104	39.9	4	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
155	KEERTHIRAM	49/M	CHEST PAIN	89	140/80	ANTEROSEPTAL	21.9	NO	NO	VEG	NO	NO	YES	NO	NO	160	19	0.7	174/29/110/190	39.7	3	MILD LV DYSFUNCTION/44.1%	DISCHARGED
156	RAMASAMY	40/M	CHEST PAIN	79	160/100	ANTERIOR WALL	23.6	YES	YES	NON VEG	NO	NO	NO	NO	YES	84	27	0.8	174/34/112/140	36.7	3	MILD LV DYSFUNCTION/47%	DISCHARGED
157	SARASWATHY	78/F	GIDDINESS	58	140/80	INFERIOR WALL	20.4	NO	NO	VEG	NO	NO	YES	NO	NO	166	39	1.3	176/32/118/134	34.4	6	MILD LV DYSFUNCTION/40%	DISCHARGED
158	GANESAN	40/M	CHEST PAIN	104	160/90	ANTERIOR WALL	24.7	YES	YES	NON VEG	NO	NO	NO	NO	YES	140	33	0.9	180/26/120/174	32.2	3	MILD LV DYSFUNCTION/48%	DISCHARGED
159	PALAPPAN	67/M	CHEST PAIN	90	130/80	ANTERIOR WALL	27.8	NO	NO	NON VEG	YES	YES	YES	NO	NO	164	29	1.2	184/30/130/120	34.9	6	MILD LV DYSFUNCTION/43%/LVH	DISCHARGED
160	SAMUEL	50/M	CHEST PAIN	106	170/90	ANTEROSEPTAL	20.8	YES	YES	NON VEG	NO	NO	NO	NO	NO	138	25	0.7	174/38/108/140	37.6	3	MILD LV DYSFUNCTION/45%	DISCHARGED
161	VELLAIMAL	70/F	GIDDINESS	68	140/90	LATERAL WALL	28.9	NO	NO	NON VEG	YES	YES	YES	NO	NO	262	40	1.6	211/36/143/162	42.8	6	MODERATE LV DYSFUNCTION/32%	DISCHARGED
162	KALIMUTHU	74/M	GIDDINESS	66	140/90	INFERIOR WALL	22.1	NO	YES	NON VEG	NO	NO	NO	NO	NO	96	41	1.2	162/48/86/136	38.7	2	MILD LV DYSFUNCTION/42%	DISCHARGED
163	KARUPPIAH	70/M	CHEST PAIN	75	160/90	INFEROPOSTERIOR WALL	21.5	YES	YES	NON VEG	NO	NO	NO	NO	NO	113	38	1.4	170/40/102/138	38.4	3	MILD LV DYSFUNCTION/47%	DISCHARGED
164	SUDALAI	72/M	GIDDINESS	56	130/80	INFERIOR WALL	21.8	YES	YES	NON VEG	NO	NO	NO	NO	NO	93	33	0.6	138/24/94/98	34.8	2	MILD LV DYSFUNCTION/47%	DISCHARGED
165	VEMBU	64/M	GIDDINESS	66	170/90	INFERIOR WALL	28.5	NO	NO	NON VEG	YES	YES	NO	YES	NO	109	36	1.2	174/34/114/130	29.8	2	NORMAL/54%/LVH	DISCHARGED
166	RAMAKRISHNAN	44/M	CHEST PAIN	90	160/100	LATERAL WALL	27.4	YES	YES	NON VEG	YES	YES	NO	NO	NO	113	44	0.9	138/18/100/102	37.7	3	MILD LV DYSFUNCTION/48%	DISCHARGED
167	ASANFATH	65/F	CHEST PAIN	82	140/90	ANTEROSEPTAL	28.4	NO	NO	NON VEG	YES	YES	NO	YES	NO	96	30	1.2	166/32/110/120	39.6	6	MILD LV DYSFUNCTION/41%/LVH	DISCHARGED
168	PONNAMBALAM	62/M	CHEST PAIN	94	180/100	ANTERIOR WALL	23.9	NO	YES	NON VEG	NO	NO	NO	YES	YES	83	18	0.9	140/28/90/110	37.6	2	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
169	MOHAMMAD	74/M	GIDDINESS	66	180/90	INFEROPOSTERIOR WALL	20.8	NO	NO	NON VEG	NO	NO	YES	YES	NO	180	41	1.4	182/34/120/140	39.7	5	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
170	KASIMANI	41/M	CHEST PAIN	104	140/90	ANTERIOR WALL	22.8	YES	YES	NON VEG	NO	NO	NO	NO	YES	90	19	0.7	138/28/90/100	39.6	5	MILD LV DYSFUNCTION/42%	DISCHARGED
171	VASANTHA	49/F	CHEST PAIN	58	150/90	INFERIOR WALL	27.9	NO	NO	VEG	YES	NO	YES	NO	NO	188	40	1.1	190/40/124/128	35.9	1	MILD LV DYSFUNCTION/46%	DISCHARGED
172	MOHD ALI	64/M	CHEST PAIN	106	150/90	ANTEROSEPTAL	24.4	NO	NO	NON VEG	NO	NO	YES	YES	NO	176	39	1.2	180/32/120/140	37.5	7	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
173	CHINNATHAI	60/F	CHEST PAIN	92	160/100	ANTEROSEPTAL	30.4	NO	NO	VEG	YES	YES	NO	YES	NO	130	39	1.3	162/30/110/110	36.7	4	MILD LV DYSFUNCTION/45%/LVH	DISCHARGED
174	MAYANDI	55/M	CHEST PAIN	62	140/80	INFEROPOSTERIOR WALL	23.9	YES	YES	NON VEG	NO	NO	NO	NO	YES	84	19	0.7	132/34/80/88	40.3	1	NORMAL/58%	DISCHARGED
175	PANDARAM	45/M	CHEST PAIN	78	130/90	LATERAL WALL	22.8	YES	YES	NON VEG	NO	NO	NO	NO	NO	122	30	1.2	138/22/96/98	30.8	2	NORMAL/56%	DISCHARGED
176	MOOKAN	55/M	GIDDINESS	58	160/100	INFERIOR WALL	24.4	YES	YES	NON VEG	NO	NO	NO	NO	NO	101	29	0.8	152/34/96/110	38.6	1	MILD LV DYSFUNCTION/48%	DISCHARGED
177	MARIMUTHU	55/M	CHEST PAIN	86	170/100	ANTERIOR WALL	21.4	YES	YES	NON VEG	NO	NO	NO	NO	YES	102	30	0.7	184/38/120/130	39.1	3	MILD LV DYSFUNCTION/44%	DISCHARGED
178	PAPPA	67/F	CHEST PAIN	82	110/70	INFERIOR WALL	18.3	NO	NO	NON VEG	NO	NO	YES	NO	YES	278	36	1.3	175/35/110/150	29.6	4	MILD LV DYSFUNCTION/46%	DISCHARGED
179	SUNDARAJ	35/M	CHEST PAIN	82	150/100	ANTEROSEPTAL	29.6	YES	YES	NON VEG	YES	YES	NO	NO	YES	272	40	1.2	180/36/110/170	43.4	2	MILD LV DYSFUNCTION/48%	DISCHARGED
180	RAMAIAH	63/M	CHEST PAIN	82	140/80	ANTEROSEPTAL	27.2	YES	YES	NON VEG	YES	NO	NO	YES	NO	190	40	1.3	190/38/115/185	40.6	3	MILD LV DYSFUNCTION/48%	DISCHARGED
181	ARPUTHAM	57/F	CHEST PAIN	86	120/80	INFERIOR WALL	24.3	NO	NO	VEG	NO	NO	NO	NO	NO	128	36	1.3	197/40/124/165	38.6	2	MILD LV DYSFUNCTION/49%	DISCHARGED
182	SETHU	50/M	CHEST PAIN	98	120/60	INFERIOR WALL	28.4	YES	YES	NON VEG	YES	NO	YES	NO	YES	220	38	1.2	192/38/119/175	30.6	2	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
183	SUDALAIMANI	52/M	CHEST PAIN	92	160/110	EXTENSIVE ANTERIOR WALL	27.9	YES	YES	NON VEG	YES	YES	NO	NO	NO	112	28	0.7	186/42/98/152	39.5	2	MODERATE LV DYSFUNCTION/33%	DISCHARGED
184	KRISHNAN	75/M	BREATHLESSNESS	92	180/100	ANTERIOR WALL	27.8	YES	NO	NON VEG	YES	YES	NO	NO	NO	123	39	1	186/38/114/170	36	5	MILD LV DYSFUNCTION/45%	DISCHARGED
185	BASKARAN	74/M	CHEST PAIN	84	170/110	ANTERIOR WALL	28.9	NO	NO	VEG	YES	YES	NO	NO	NO	128	38	1.2	186/38/114/170	40.5	4	MILD LV DYSFUNCTION/48%	DISCHARGED
186	BEEVI	55/F	CHEST PAIN	68	160/100	INFERIOR WALL	26.7	NO	NO	NON VEG	YES	YES	NO	YES	YES	118	30	1.2	130/22/86/110	33.1	1	NORMAL/54%/LVH	DISCHARGED
187	SUDALAIMADAN	65/M	CHEST PAIN	90	170/100	ANTERIOR WALL	24.2	NO	NO	NON VEG	NO	NO	NO	YES	NO	130	46	0.9	164/20/106/190	39.6	5	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
188	CHELLAIAH	55/M	CHEST PAIN	58	180/100	INFEROPOSTERIOR WALL	26.9	YES	YES	NON VEG	YES	YES	NO	YES	NO	114	41	1.2	174/40/98/186	30.6	1	NORMAL/56%/LVH	DISCHARGED
189	GOMATHY	70/F	CHEST PAIN	86	150/90	ANTEROSEPTAL	29.8	NO	NO	VEG	YES	YES	NO	YES	YES	116	39	0.9	174/38/100/180	39.5	6	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
190	MUPPIDATHY	60/F	CHEST PAIN	64	180/100	INFERIOR WALL	27.6	NO	NO	NON VEG	YES	YES	NO	YES	NO	78	30	1	173/33/110/150	33.5	1	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
191	LAKSHMI	56/F	CHEST PAIN	76	140/80	ANTEROSEPTAL	28.4	NO	NO	VEG	YES	YES	YES	NO	NO	166	39	1.3	133/30/67/180	32.1	3	MILD LV DYSFUNCTION/43%	DISCHARGED
192	SUBBIAH	65/M	CHEST PAIN	90	160/80	ANTERIOR WALL	27.9	NO	NO	VEG	YES	YES	YES	NO	NO	133	33	0.7	246/42/168/180	39.2	4	MILD LV DYSFUNCTION/48%	DISCHARGED
193	MARIAMMAL	65/F	CHEST PAIN	56	160/90	INFERIOR WALL	20.8	NO	NO	VEG	NO	NO	NO	YES	NO	112	40	1.6	186/36/120/150	35.8	3	MILD LV DYSFUNCTION/44%/LVH	DISCHARGED
194	PARVATHY	75/F	GIDDINESS	64	170/100	INFERIOR WALL	24.8	NO	NO	VEG	NO	NO	YES	YES	NO	180	41	1.2	184/34/118/160	39.6	6	MILD LV DYSFUNCTION/48%/LVH	DISCHARGED
195	RAMAPANDIAN	75/M	BREATHLESSNESS	104	180/100	ANTERIOR WALL	22.9	YES	NO	NON VEG	NO	NO	NO	YES	NO	134	38	1.2	212/30/150/160	42.2	10	MODERATE LV DYSFUNCTION/34%/LVH	DISCHARGED
196	ARULDOSS	58/M	CHEST PAIN	68	140/90	INFEROLATERAL WALL	23.7	YES	YES	NON VEG	NO	NO	YES	NO	NO	190	16	0.7	155/37/91/135	39.6	3	MILD LV DYSFUNCTION/46%	DISCHARGED
197	SUBBAMAL	65/F	CHEST PAIN	66	150/80	INFERIOR WALL	28.4	NO	NO	VEG	YES	YES	YES	NO	NO	217	41	1.1	171/33/120/137	34.4	6	MILD LV DYSFUNCTION/45%	DISCHARGED
198	SUDALAIMUTHU	57/M	CHEST PAIN	98	170/100	EXTENSIVE ANTERIOR WALL	23.5	YES	YES	NON VEG	NO	NO	YES	YES	YES	184	38	1.4	194/42/126/130	38.6	4	MILD LV DYSFUNCTION/43%/LVH	DISCHARGED
199	THANGAVEL	73/M	GIDDINESS	64	160/100	LATERAL WALL	20.6	YES	NO	NON VEG	NO	NO	YES	YES	NO	184	38	1.2	214/38/140/184	35.5	5	MILD LV DYSFUNCTION/42%/LVH	DISCHARGED
200	SELVAMUTHU	43/M	CHEST PAIN	70	140/80	INFERIOR/RV MI	21.7	YES	YES	NON VEG	NO	NO	NO	NO	NO	89	26	0.8	166/36/96/170	30.3	2	NORMAL/58%	DISCHARGED

ANNEXURE-3

KEY TO MASTER CHART

Sex:

M- Male, F-Female

Presenting Complaints:

Chest Pain, Giddiness, Palpitation, Breathlessness

MI Type:

Anterior Wall, Anteroseptal Wall, Anterolateral Wall, Extensive Anterior Wall, Inferior Wall, Inferoposterior Wall, Lateral Wall, Inferior/Right Ventricular Wall

BMI:

<18.5- Underweight, 18.5 to 24.9- Normal weight, 25 to 29.9-Overweight, 30 and above – Obese

Food Habits:

Vegetarian, Non-Vegetarian

RiskFactors:(Smoking to Family History of CAD)

YES, NO

Echo Study:

Normal Left Ventricular function,Mild Left Ventricular Dysfunction, Moderate Left Ventricular Dysfunction, Severe Left Ventricular Dysfunction

Outcome:

Discharge, Death.

ANNEXURE-4

ABBREVIATIONS

BP- Blood Pressure

CAD- Coronary Artery Disease

CHD-Coronary Heart Disease

CVD-Cardio Vascular Disease

ECG- Electrocardiogram

LMCA-Left Main Coronary Artery

LAD-Left Anterior Descending Artery

LCX-Left Circumflex Artery

RCA-Right Coronary Artery

LV - Left Ventricle

MI- Myocardial Infarction

TIMI-Thrombolysis in Myocardial Infarction

STEMI- ST Elevation Myocardial Infarction

CPK-MB – CreatininePhospho Kinase - MB Isoenzyme

DHA-Docosa Hexaenoic Acid

EPA- Eicosa Pentaenoic Acid

ALA-Alpha Linoleic Acid

LA- Linoleic Acid

HDL- High Density Lipoprotein

LDL-Low Density Lipoprotein

VLDL- Very Low Density Lipoprotein

TG-Triglycerides

SFA- Saturated Fatty Acids

MUFA- MonoUnsaturated Fatty Acids

PUFA-Poly Unsaturated Fatty Acids

TFA- Trans Fatty Acids

WHO-World Health Organisation